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Policy

The U.S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article, in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

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SPECIAL ARTICLE

THE NAVY HOSPITAL CORPSMAN AS A TRAINED HEALTH CARE ASSISTANT*

CAPT Roger Stevenson, MC, USN, Director, Professional Division, Bureau of Medicine and Surgery, Navy Department.

Numerous training programs are appearing across the country for the preparation of various categories of health care assistants. This seems to indicate recognition of the critical need to augment acutely short professional manpower resources in the health care and related fields. For many years the Navy, along with our other Federal services, has recognized and effectively met the requirement to supplement the services of scarce personnel with non-professional and well trained assistants. In meeting this requirement, the Navy has trained and utilized personnel who, following completion of active military service, can continue to contribute to increasing the total pool of available health manpower. It is a privilege for me to have this opportunity of sharing with you some of the experiences which we have had in the training and utilization of health care assistants.

To go back in history, for just a few moments, the records of the Navy during the Revolutionary War indicate that there was probably very little effort to provide for the care of the sick and injured either afloat or ashore. Available naval medical service was limited to the individual capacity of the ship's surgeon or the surgeon's mate. The appointment of medical personnel was in most cases a matter of expediency and was made on the spot, without much formality, by the captain of the ship, on his own authority, and only for the duration of one cruise.

During this era there were seemingly no enlisted men trained in the care of the sick and injured.

As the assistant surgeon on the ship gradually limited his field to the strictly professional aspects of his functions, it became increasingly necessary to delegate to the sick bay attendant such minor duties as nursing, dispensing of medication, first aid, and clerical work as his experience, knowledge and training would permit. Under these circumstances the sick bay attendant assumed a degree of importance and gradually became a trained assistant to the medical officer.

By 1866 the standing of the original loblolly boys (the designation for the first non-professional health care assistants) had been advanced considerably. The need of trained personnel to assist the medical officer was now more apparent and generally recognized in the Navy. The Hospital Corps, as an organized unit of the Medical Department of the Navy was created by an act of Congress, approved 17 June 1898, at the onset of the Spanish American War. The establishment of the Hospital Corps represented the fruition of a century long struggle to establish the non-commissioned personnel of the medical department on a sound footing, and the beginning of a new era of progress that has culminated in outstanding medical service for Navy and Marine Corps personnel.

In 1902 the first Hospital Corps Training School was established in Norfolk, Virginia, with the objective of providing uniform and systematic training for new hospital corps personnel coming into the Navy. This facility was a forerunner of other schools that have been established from time to time to provide the Medical Department with a corps of well trained, competent personnel for every emergency.

By 1950, prior to the outbreak of hostilities in Korea, there were more than 40 separate training courses in the medical and dental technical specialties available to personnel of the Hospital Corps. The scope of instruction in these schools embraced administrative procedures and the technical aspects of medical and dental sciences.

Navy Hospital Corpsmen now serve around the world in hospitals and ships, often on duty independent of a medical officer, thus bearing the full responsibility for the medical care of their shipmates, maintaining the health of the Navy, rendering first aid, and caring for the sick with a competence that has earned the respect of all.

Their methods of medical management are constantly reviewed and revised to reflect the latest trend in treatment. Since 1958, for example, they have received special and intensive instruction in

* Presented at the American Hospital Association Annual Meeting, Federal Panel Instructional Session, Chicago, Illinois, 19 August 1969.

the management of mass casualties which might result from a thermo-nuclear, biological or chemical warfare attack or from natural disaster.

Every corpsman receives initially the basic 14-week Hospital Corps School course of instruction. He is then generally assigned to a hospital ward where he serves as an extension of the nurse's arm and becomes quite proficient in the bedside nursing care of patients. After the man has had several years experience he can attend a 20-week course to qualify as an advanced service technician. He receives training in advanced principles and techniques of patient care, first aid and emergency procedures, preventive medicine, and medical administration, with emphasis on tentative diagnosis and acquiring the skills necessary for assignment to duty independent of direct medical officer supervision, his only contact with a medical officer being by radio. This course also includes instruction in materia medica, pharmacology, and routine laboratory techniques and procedures.

The Navy has courses, ranging from 26 to 52 weeks in duration, for training many other types of health care assistants.

The tables below list the many fields in which training as a health care assistant is available for the Navy corpsman. Table I—The Medical Field Service Technician has had training along much the same lines as the Advanced General Service Technician. He can administer life saving first aid, do minor suturing, and generally has had experience in triage procedures. These two can serve very adequately as assistants in emergency rooms, taking and recording histories and doing certain parts of the physical examination.

Table I

Medical Field Service Technician
Advanced General Service Technician

Table II lists a group of technicians who can be of particular assistance to the physician in various special areas of the hospital. Our operating room, eye, ear, nose and throat, urological, and orthopedic cast room technicians are especially proficient at assisting the physician in those special areas. I am sure the civilian community can always use more of the other technicians and assistants listed in this table. These people are equally proficient in their respective fields of work.

Table III lists another group of technicians who are not necessarily useful around the hospital but

Table II

Nuclear Medicine Technician
Cardiopulmonary Technician
Clinical Laboratory Assistant
Clinical Chemistry Technician
Radioisotopes Technician
Clinical Laboratory Technician
X-Ray Technician
Electroencephalography Technician
Electrocardiograph & Basal Metabolism Technician
Operating Room Technician
Eye, Ear, Nose, and Throat Technician
Neuropsychiatry Technician
Urological Technician
Orthopedic Cast Room Technician
Dermatology Technician

Table III

Special Operations Technician
Nuclear Submarine Medicine Technician
Submarine Medicine Technician
Aviation Medicine Technician
Aviation Physiology Technician
Tissue Culture Technician
Preventive Medicine Technician
Medical Administrative Technician
Optical (General) Technician
Optical (Laboratory) Technician
Photography Technician
Pharmacy Technician
Physical & Occupational Therapy Technician
Medical Deep Sea Diving Technician
Medical Illustrating Technician
Medical Repair Technician

can be of great help in industrial and preventive medicine situations. The tissue culture, medical administrative, photography, physical and occupational therapy and medical illustrating technicians could, of course, be of assistance in some hospitals, as could the medical repair technician. The last named are qualified to maintain and repair almost any piece of medical equipment, including electronic gear.

Table IV is added for completeness. We do train health care assistants for dentists as well as physicians!

Another area which the Navy began investigating in late 1967 was the use of trained ward managers. One of the primary objectives of the introduction of

Table IV

Advanced General Dental Technician
Field Service Dental Technician
Clinical Laboratory Dental Technician
Research Assistant
*Administrative Dental Technician
Dental Equipment Repair Technician
Advanced Prosthetic Technician
Maxillofacial Prosthetic Technician

*Same course of instruction as Medical Administrative Technician

the ward manager system was to improve the utilization of scarce nursing manpower. The ward manager was introduced as a person to whom all of the non-nursing duties and responsibilities could be assigned so that the nurse could return to patient care. In order to truly relieve the nurse of the responsibility for non-nursing activities, the ward manager was made responsible to hospital administration rather than nursing service.

His duties fall into four broad categories: (1) maintenance of the environment; (2) maintenance of materials; (3) maintenance of administrative records, preparation and submission of administrative reports; and (4) coordination of hospital services for patient care.

Hospital corpsmen are prepared to function as ward managers by formalized program of instruction approximately 14 weeks in duration. The training program includes four weeks' didactic instruction and ten weeks of concurrent supervised practice and instruction. The research results have demonstrated that the ward manager truly does relieve nursing personnel from administrative tasks, thus freeing them to return to patient care. We feel that many of our corpsmen can be utilized in the health care field after leaving the service. All of these technicians must have at least a high school equivalency in education and some of them are required to have didactic or practical training that amounts to at least two years of college. The Navy is investigating the possibility of tying our technician training programs in with an associate degree program in local colleges. Some of our technicians can and do qualify for accreditation by civilian agencies. Among these are the x-ray and radioisotope technicians, laboratory technicians, and optician technicians. Many others, however, are unfortunately lost to the health care field once they return to civilian life.

MEDICAL ARTICLES

SEPARATION PROBLEMS IN MILITARY WIVES

Houston MacIntosh, MD, Amer J Psychiat 125(2):260–265, August 1968.

Sixty-three military wives experiencing psychiatric disturbances while separated from their husbands for military reasons are compared with 113 wives not separated but needing psychiatric help. Some important psychodynamics of separation are discussed, and a statistical comparison of the two groups is made. The subject is becoming more important to nonmilitary psychiatrists in view of the expanded military dependents' health care program.

Penelope, the wife of Ulysses, was forced to cope with many difficulties during her husband's absence during the Trojan War. Today military psychiatrists and increasing numbers of civilian psychiatrists are faced with modern Penelopes who are unsuccessful in adapting to their husbands' absence.

During World War II Helene Deutsch reported that a number of "war neuroses" in wives had been observed at the psychiatric clinic of the Boston Psychoanalytic Institute. By this she meant neurotic disturbance in women precipitated by separation from their husbands because of military service.

Recent psychiatric literature has made passing reference to psychiatric disturbance in military wives precipitated or aggravated by separation from their

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The opinions expressed in this paper are Dr. MacIntosh's own and do not necessarily reflect those of the Department of Defense or any of its branches.

The author wishes to thank Nathan Simon, M.D., for his advice in the preparation of this paper.

husbands because of military duties. In general, however, the military psychiatric literature has not focused on this important clinical problem. Because of the recent expansion of military dependents' health care to cover outpatient psychiatric treatment, and the continuing shortage of military psychiatrists, this clinical problem is becoming of increasing interest to civilian psychiatrists.

This paper focuses on the military wife who is separated from her husband, and the psychiatric symptoms which are precipitated or aggravated by the separation. An attempt is made to determine the magnitude of this problem. Sixty-three women who developed symptoms are compared with a control group of 113. Some of the dynamics of separation psychopathology are discussed.

Method

The data for the present study were collected while the author was director of a psychiatric outpatient clinic in an Air Force general hospital. The clinic is the only military psychiatric facility servicing a large midwestern metropolitan area. All civilian dependents are screened by a physician before they are seen for psychiatric consultation.

The psychiatric records for all married women with husbands on active duty were reviewed for the 18-month period from August 1965 to February 1967. All subjects were seen by the author for a diagnostic psychiatric interview. Additional data were available on some who were subsequently seen in individual or group psychotherapy.

The women were divided into two groups for the purpose of this study. The first group, designated as "separated," consisted of those who displayed psychiatric symptoms precipitated or aggravated by a current or impending separation from their husbands. Also included in this group were women who were first seen after the return of their husbands and displayed symptoms precipitated or aggravated by the end of an extended separation; these women may also have had symptoms during the separation. "Separation" is defined as the husband being physically absent from the household for at least two months. (In most cases a husband's "unaccompanied tour" lasts for one year.) A few cases were included in which briefer but frequent and repeated separations occurred over a period of several months. The other group, those "not separated," was composed of women who, at the time of their referral, did not fulfill the above criteria.

Results

The results are summarized in tables 1, 2, and 3. The separated wives comprised 36 percent of the total. They were significantly younger and less well-educated ($p = .02$). Army wives were significantly over-represented ($p = .01$). There was a trend toward fewer Air Force wives ($p = .12$), more enlisted wives ($p = .18$), and fewer officers' wives ($p = .25$). There were significantly fewer foreign-born wives ($p = .01$). There was a slight trend toward more neurotic reactions ($p = .31$) and fewer psychotic reactions ($p = .25$). Number of children, racial distribution, and suicide attempts were similar in both groups.

Most of the separated wives developed symptoms during the actual absence of their husbands. About a third had husbands serving in a war zone. Thirty percent requested that their husbands be returned or given a change of assignment so they could be together. This request was statistically significantly less, however, if the husband was serving in a war zone. The majority of the separated group were living with or near parents or relatives.

Discussion

The data indicate that a significant number of military wives who are referred to a psychiatrist

TABLE 1. *General Comparison of Military Wives Referred to a Psychiatric Clinic*

Variable	Separated* from Husbands (N=63)		Not Separated from Husbands (N=113)	
	Mean	Range	Mean	Range
Age in number of years**	30.2	17–43	33.8	18–57
Education in number of years**	11.3	6–16	12.1	6–16
Number of children	2.7	0–6	2.5	0–9

*Husband physically absent from household because of military duties.
**Difference of the means, 2.5 times the standard error; p=.02.

TABLE 2. *Selected Comparison of Military Wives Referred to a Psychiatric Clinic*

Variable	Separated* from Husbands (N=63)		Not Separated from Husbands (N=113)	
	Number	Percent	Number	Percent
Husband's branch of service				
Air Force	42	67	98	87
Army**	17	27	9	8
Other	4	6	6	5
Husband's rank				
Enlisted	15	24	17	15
NCO	35	56	60	53
Officer	13	21	36	32
Foreign-born**	4	6	23	20
Race				
Caucasian	59	94	105	93
Negro	3	5	7	6
Other	1	1	1	1
Attempted suicide	5	8	10	9
Diagnosis				
Psychoneurotic reaction	29	46	40	35
Personality disorder	23	37	45	40
Psychotic reaction	8	13	22	19
Other	3	5	6	5

*Husband physically absent from household because of military duties.
**p=.01 by X^2 test.

TABLE 3. *Selected Characteristics of Military Wives Separated* from Their Husbands and Referred to a Psychiatric Clinic*

Variable	Number (N=63)	Percent
Time of referral		
Before husband's absence	15	24
During husband's absence	37	59
After husband's absence	11	17
Place of residence (actual or anticipated)		
Self	23	37
Parents	26	41
In-laws	5	8
Other relatives	2	3
Unknown or undecided	7	11
Husband serving in war zone (actual or anticipated)	23	37
Change of assignment or return of husband requested		
Husband in war zone (N=23)**	3	14
Husband not in war zone (N=40)	16	40
Total (N=63)	19	30

*Husband physically absent from household because of military duties.
**p=.01 by X^2 test.

have symptoms directly related to separation from the husband because of military duties. The total number of military wives developing symptoms in response to this particular social stress is speculative but is most likely of public health significance.

Lemkau has pointed to the value of collecting and interpreting service statistics to determine incidence of mental disorder and to plan for mental health services. He has also indicated the need for research with special risk groups. Military wives would certainly constitute a special risk group regarding separation problems.

The separated wives were younger; this suggests that the older women had previously found ways of coping with separation. Separation, for a military wife, may be a developmental task which is more difficult early in life and becomes easier with practice. The factor of less education suggests poorer ego development, less intellectual capacity, and lower frustration tolerance. The fact that there were more Army wives in the separated group is not explained by attempts at correlation with other variables in the study. A possible explanation is the fact that the Air Force is an all-volunteer service whereas the Army is not.

The meaning of the husband's absence to his wife is probably important in determining her response. This may account for the Army-Air Force difference as well as the enlisted man-officer difference. Many officers feel that an assignment to Vietnam will further their careers. Wives frequently identify with the status of their husbands ("She wears her husband's rank."), which may provide sufficient narcissistic gratification to allow them to endure the other difficulties of separation. This is less likely to be the case for the wife of an Army draftee. If a woman has significant masculine strivings and competitive feelings toward her husband, an absence which increases his status may provoke symptoms, as is illustrated by the following case:

Case 1. Mrs. A. is a 32-year-old efficient, aggressive graduate nurse, the mother of four boys. Two years prior to referral she had reluctantly undergone a hysterectomy which she felt had been forced on her by her husband and her doctor, who had warned her that if it was not done "Some day you'll come into the hospital bleeding and in shock." After the operation she was chronically depressed and irritable with her husband, but was able to function moderately well. Prior to her referral her husband, a medical corpsman, was sent to the noncommissioned officers' academy at another base, which was an honor for him and helpful to his career. When he returned she provoked him, and they argued continuously. A few days later she cut her throat. She was discovered by her "favorite" son who brought her to the hospital actually "bleeding and in shock."

There were significantly fewer foreign-born wives in the separated group. Most of the foreign-born wives seen were struggling with problems related to separation from their parents and adjusting to a new culture. A possible explanation for the difference in the separated and nonseparated groups is that if a foreign-born wife has the capacity to adapt to separation from her parents, she is more probably able to adapt to separation from her husband.

In discussing military wives separated from their husbands, Helene Deutsch wrote:

"What is in question is not the women's erotic longing, or the absence of the father as a family supporter, but the motherliness of the husband, without whose active help the wife cannot function. In the life histories of such mothers, one always discovers a strong infantile dependence upon their own mothers that has been transferred to their husbands."

The women studied here frequently confirm Deutsch's observation and emphasize the central importance of dependency conflicts.

Case 2. Mrs. B. is a 30-year-old mother of three who was referred by a neurologist. She complained of nausea, vomiting, and headaches "like my head has been split open." The symptoms had been present since her husband left for a noncombat area. She had never been separated from him in 11 years, and they had always done everything together. She had had the feeling "Other husbands got sent away but mine won't." She begged for his return.

Her mother had invited her to live with her during her husband's absence. Because of her headaches, Mrs. B. was spending three-fourths of her time in bed, and her mother had to quit work in order to care for her and the grandchildren. As a child Mrs. B. had felt that her mother was cold and distant to her but was affectionate with her siblings.

Some military psychiatrists feel that women find support by return to a "familiar family milieu and relatives." My data indicate that this is not always the case. The majority of the separated group were living with or near relatives. Since women with unresolved dependency and oedipal conflicts may deal with these issues by leaving home at adolescence and marrying, the husband's absence and returning to live with parents presents an opportunity for regression, with consequent reactivation of unresolved conflicts. The following case illustrates this point. The case is also interesting because it illustrates the role played by the husband when he projects his own dependency needs onto his wife.

Case 3. Mrs. C. is a 24-year-old mother of three. She complained of anxiety attacks relieved only by her husband's being with her. These were particularly severe when she was shopping for groceries or walking in open spaces, and when visitors came to their home. The symptoms had been present for a year since her husband's return from a noncombat area.

She had had a close, affectionate relationship with her father who had died suddenly when she was 14 years old. Her mother had married an Air Force NCO a year later. Mrs. C. married a young man in the Air Force at age 17 and left home. Things went well for five years until her husband received a one-year remote assignment. Her mother insisted she come to live nearby during her husband's absence but Mrs. C. felt she could manage on her own. Her husband sided with her mother, and under the pressure she agreed.

For the first six months she was lonely and occasionally depressed but functioned adequately. One night when she was visiting her parents' home and her mother was out, her stepfather kissed her. She felt sexually excited but overcome with guilt and panic. She began to vomit the next morning and continued to do so every morning until her husband returned. Her mother insisted she move into the same house to take better care of her, and Mrs. C. felt she had to comply. Her symptoms became more severe but because of her guilt she was unable to talk freely with her mother or her doctors.

Some women act out sexual impulses when their husbands are away and this may produce family turmoil later on.

Case 4. Mrs. D. is a 30-year-old mother of four. She complained of depression and anxiety, fought constantly with her husband, and threatened suicide. When her husband had been overseas she had had an affair and became pregnant. When she later joined him she offered to give the baby up for adoption but her husband refused. He tried to get the other man court-martialed but was unsuccessful as his wife would not cooperate. They returned to the same base where the man was stationed; the husband became paranoid regarding this man and finally succeeded in getting him transferred shortly before Mrs. D.'s consultation.

They fought constantly over sex. Mrs. D. felt her husband had wanted intercourse too often since their return. She disliked sex with her husband and feared pregnancy. She started taking birth control pills, but then could not enjoy intercourse because she knew she could not become pregnant. This behavior enraged her husband because she could "enjoy it with the other man," but not with him.

Anna Freud has described a form of separation anxiety which occurs in children when they are required to leave their parents to go to school. She writes:

"The conflict between love and hate of the parents can be tolerated by the child only in their reassuring presence. In their absence, the hostile side of the ambivalence assumes frightening proportions, and the ambivalently loved figures of the parents are clung to so as to save them from the child's own death wishes, aggressive fantasies, etc."

A similar situation can develop in a woman who has transferred ambivalent attitudes toward her parent onto her husband and then is forced to be separated from him. If a husband is placed in a situation such as combat, where his life is actually in danger, one might expect unusual anxiety. This was noted in some of the women in this study. However, only 37 percent had husbands in combat zones and they, interestingly, requested the husband's return significantly less often than women whose husbands were in a noncombat area.

Case 5. Mrs. E. is a 26-year-old mother of three who complained of depression, feelings of worthlessness, weight loss, inability to take care of her children, and an intense delusional fear that she had cancer and was going to die. These feelings had been present in the past but had become much worse five months earlier when her husband was sent to another base for temporary duty. Although they had been married for eight years she had hardly ever been separated from him. Whenever they were apart she was anxious and had obsessional thoughts that he would die.

Mrs. E. was the third child in a family of seven. Her parents were always "sick" when she was a child and there were constant family crises and turmoil. She felt that because of her mother's sickness she was always being given responsibilities she did not want. She recalled being furious and accusing her mother of faking illness. She recalled intense anxiety whenever separated from her mother as a child, being afraid her mother would die. She had similar but less intense feelings regarding her father and siblings. Six years before her referral, her mother was successfully operated on for breast cancer. Mrs. E. had been moderately hypochondriacal since then.

Should the psychiatrist attempt to arrange the return of a serviceman whose wife is experiencing psychiatric disturbance related to his absence? The author feels that in most cases this is neither desirable nor necessary. It offers the wife an opportunity for regression and secondary gain by assuming a sick role; moreover it is a denial of the social reality of being a military wife. The expense of time and money to the government is obvious.

I feel, however, that these women should be given the opportunity for adequate psychiatric treatment. The author's experience in treating a few of the women in this study suggests that most women will be able to adapt if adequate treatment is provided. In those women who cannot tolerate this approach there is usually poor frustration tolerance and severe character pathology. In these cases I advise that the husband not reenlist and that he find a career in which he can be home more regularly.

(The references may be seen in the original article.)

ANTICOAGULANT THERAPY—PRACTICAL MANAGEMENT

I. S. Wright, MD, New York, N. Y., Amer Heart J 77(2):280–286, February 1969.*

This year marks the thirtieth anniversary of the first administration of anticoagulants to a patient in the United States. Dr. Charles Best brought his supply of the available purified heparin from Toronto to New York and he and I administered this jointly to a patient who was suffering from intractable migratory thrombophlebitis of many months duration. During the period of exploration, we have made or reviewed most of the possible mistakes and variations in the forms of anticoagulant therapy which have been in common use. There are two essential steps in treatment. The first is carefully selecting the patient who will receive it. The second is choosing the appropriate technique of administration, for anticoagulants can be satisfactorily administered by several techniques. Too frequently in clinical practice, the dosage has been either inadequate, inviting additional thromboembolic complications, or excessive, encouraging the development of hemorrhagic complications. Errors in dosage have been common in the case of individual patients, and have invalidated some large studies otherwise well designed to evaluate their use.

Like a football team, if the individual participants (in this case physicians) within a single institution use too many variations from a reasonably standardized procedure, the chance for error and complications increases. This article will deal primarily with the technique and practical aspects of anticoagulant therapy, but first some general statements must be made regarding selection of patients.

Selection of Patients

The only logical reasons for the use of anticoagulant therapy are to prevent or to treat thromboembolic complications. Thromboembolism plays a major role in determining the degree of morbidity and the mortality of the majority of people who die in their middle or latter years.

Therefore, use anticoagulants to: (1) prevent the formation of thrombi in conditions known to be favorable to their development; (2) permit the dissolution of sludge or early soft thrombus by allowing the fibrinolytic enzymes of the blood to become more active without the usual inhibiting forces; (3) prevent the mother clot whether intracardial or intravascular from propagation and embolization; and (4) discourage the propagation of clots within vessels from blocking off additional branches of these vessels.

The exact indications in terms of heart disease, cerebral vascular disease, peripheral arterial and venous disease, the problems of pulmonary embolism, and many other manifestations are not within the scope of this presentation. There are certain absolute contraindications, namely, the presence or immediate past history of severe hemorrhage, recent central nervous system surgery, hemorrhagic blood dyscrasias of significance, prostatectomy, and pericarditis, but not including the friction rub associated with myocardial infarction. Relative contraindications suggesting a need for caution include a history of old gastrointestinal ulcers or colitis, now inactive, hypertension in excess of 180/110, polycythemia of a marked degree especially if hemorrhagic manifestations are significant, and the presence of renal or liver disease. Treatment with anticoagulants should never be considered as routine. Each case should be decided on the basis of the existing and probable indications and risks.

For example, a slowly developing thrombophlebitis in the lower calf may usually be treated with oral anticoagulants. However, if a patient has had one or more recent pulmonary emboli it is preferable to initiate the treatment with heparin changing to oral anticoagulants later after therapeutic activity has been achieved. With myocardial infarction there is a difference of opinion. Some physicians prefer to initiate treatment with heparin, and at the same time start a coumarin. The heparin is then discontinued as the prothrombin time (activity) reaches a therapeutic level. Others are content, for so-called "good risk" cases, to start with oral anticoagulants. In view of the difficulty in predicting which cases will develop further thromboembolic complications, the recommended procedure is to use heparin from the beginning.

With different thromboembolic cerebral vascular states there is also a justifiable modification in treatment. Heparin is recommended to initiate the treatment of patients with transient ischemic attacks or progressive strokes. With a cerebral embolus, where

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there is diapedesis and more edema, there is a greater tendency for hemorrhage (*in the brain tissue*). During the early hours after embolization, therefore, it is considered wise not to use heparin but to wait for three or four days for stabilization of the local condition before the administration of oral anticoagulants.

The question of their use in the treatment of completed strokes is still subject to some difference of opinion, and this controversy is not within the scope of this paper. If, as the evidence suggests, they are to be used for the prevention of future thromboembolism, oral anticoagulants are adequate.

In the presence of recurrent thromboembolism, decisions must be made about whether the treatment is to be short-term or long-term, and if long-term, how long. In some patients where repeated cessation of treatment has resulted in new thromboembolism, they may be used for years.

Control and Training

There is an absolute need for accuracy in detail. This therapy demands a disciplined, experienced control. Unless a physician is prepared to deliver this type of service, he should request help from a trained team. Surveys have revealed that in hospitals where there is no well-organized anticoagulant team or where the physicians have no special interest in this therapy, the therapeutic response has been unsatisfactory and the complication rate has been high. Correction of this situation results in marked reduction in the incidence of complications.

Studies reported by Dr. George Mayer of Queens University in Kingston, Ontario, revealed that when the physicians of that institution first started to use anticoagulants the incidence of error was considerably higher than later on in their training. When handled by inexperienced physicians four times as many errors were made in treatment of outpatients as in treatment of hospitalized patients. Such errors resulted in inefficiency of anticoagulant therapy when the house staff was beginning training. As their efficiency increased during five months, there was a 67 percent decrease in errors in hospitalized patients and an 87 percent decrease in errors in outpatients. The organization of an anticoagulant unit and the supervision by an expert permitted the detection and correction of 80 to 95 percent of the potential errors. This confirms the general experience at the New York Hospital where a special team established the first anticoagulant clinic and has continued to provide a consultant service to the hospital staff. A meeting of the resident staff is held at the beginning of each new service year when certain guidelines are outlined for the techniques to be used. A liaison is then established between the anticoagulant service group who attend this session and the resident staff. This is necessitated by the fact that the resident staff come from many medical schools where a variety of techniques are used. Regrettably, some arrive with no previous training in this field.

Selection and Administration of the Anticoagulant

There is now a considerable choice of anticoagulants which can be used. Heparin is most widely used as the parenteral anticoagulant, although warfarin may be used for this purpose. Heparin can be administered subcutaneously, intramuscularly, and intravenously. There have been preparations of long-acting heparin for intramuscular use in which a variety of vehicles have been used.

Continuous intravenous heparin may be well controlled by the frequent use of Lee-White clotting time tests. This requires constant supervision as under postoperative situations. The long-term use of intravenous heparin is not practical and indeed is subject to increasing error as the resident staff becomes fatigued with the supervision of the clotting time tests over a period of days. Subcutaneous or intramuscular injections of heparin sodium are now used more frequently. They are administered at intervals of six to eight hours and this usually proves to be satisfactory. The dosage depends in part on the acuteness of the situation. In general, the initial dose is 10,000 units of heparin sodium given subcutaneously. The clotting time should be tested before the first injection for control purposes and at the end of eight hours. The clotting time should return to about twice normal before administering a subsequent dose. The clotting time may reach twice normal at eight hours, ten hours, or twelve hours. This should be the guide for the timing of the next injection of 10,000 units. Once the time interval has been determined for a specific patient, it is not necessary to check the clotting time more than once a day before the administration of a subsequent dose. This method appears to be satisfactory and has been widely used in this country as well as in Europe.

There is a greater selection of oral anticoagulants. The first was Dicumarol. The initial or loading dose is 200 to 300 mg. The second dose, administered the next day, should be 150 mg, and thereafter 50

TABLE 1. *Anticoagulant Drugs for Oral Use*

Drug	Loading Dose (mg.) (First 24–48 hr.)	Maintenance Dose (mg.)	Time to Produce Therapeutic Levels (hr.)
Coumarin compounds			
Cyclocoumarol	125–200	12.5–50	36–72
Bishydroxycoumarin	200–300	25–150	36–72
Ethyl biscoumacetate	1,800–2,400	150–900	18–36
Nicoumalone	36–52	2–12	24–42
Phenprocoumon	18–30	0.75–6	30–48
Sodium warfarin	25–30	2.5–10	36–48
Indandione compounds			
Anisindione	800–900	75–100	36–60
Diphenadione	30–45	3–5	48–60
Phenindione	200–300	25–200	36–48

or 75 mg may be given daily depending upon the prothrombin time. There are patients who require more or less than these suggested doses. The most widely used prothrombin time tests are based on the Quick one stage method. Although other tests have been advocated, the Quick test is satisfactory for practical use. The prothrombin time should be kept at about two times the normal control during the acute phases of the thromboembolic disease. For example, if the control time is 12 seconds, the desired level should be 24 seconds, plus or minus 2 seconds. This results in a percentage of prothrombin activity of approximately 20 percent. For long-term therapy it is desirable that the prothrombin time be at a level approximating 1½ to 2 times normal.

Other oral anticoagulants which have been used widely both here and abroad include: cyclocoumarol, bishydroxycoumarin, ethyl biscoumacetate, phenprocoumon, nicoumalone, and sodium warfarin. In the United States, sodium warfarin (Coumadin) is used most widely. The preferred loading dose is 25 to 30 mg and the maintenance dose averages between 2.5 and 10 mg per day. The effort should be to keep the prothrombin time within the limits above mentioned. Some patients require only 2.5 mg every other day and others require much larger doses to secure adequate therapeutic levels. Indandione preparations have been used more widely in Europe than in this country. They have essentially the same action and effect on the prothrombin time, but reports suggest a greater incidence of hypersensitivity. They also color the urine orange so the ambulatory patient is less able to recognize minor urinary bleeding. Table I lists some statistics on the oral anticoagulants that are presently available.

Studies with other drugs include thrombolytic agents involving streptokinase and urokinase. More recently, the Maylayan pit viper venom (Fraction 6) (Arvin) has been studied in England. These agents are still experimental and are not yet ready for general clinical use. Although of great scientific interest, they will not be discussed further in this paper.

Interactions With Other Drugs

An important consideration in this therapy rests in the effect of other drugs used simultaneously upon the prothrombin time. This has been well summarized in the *Medical Letter on Drugs and Therapeutics* (Vol. 9, Dec. 1, 1967). Both potentiation and inhibition of anticoagulant activity has been encountered. Some drugs when used concurrently increase microsomal enzyme activity in the liver which in turn increases metabolic degradation of the coumarins. This reduces their anticoagulant effect. When such concurrent drugs are discontinued, one can expect an increase in the prothrombin time, sometimes to dangerous levels, unless the anticoagulant dosage is reduced. These drugs include: phenobarbital, chloral hydrate, glutethimide (Doriden), meprobamate, griseofulvin, and haloperidol (Haldol). Potentiating effects may also occur with drugs that inhibit the metabolic degradation of coumarins such as phenyramidol (Analexin). On the other hand, some drugs produce potentiating effects by displacing the anticoagulant from protein-binding sites in the plasma, thus increasing the peak concentration of free coumarins. These include: phenylbutazone (Butazolidin), oxyphenbutazone (Tandearil), diphenylhydantoin (Dilantin), and the most commonly used salicylates.

Sulfisoxazole (Gantrisin, and other brands), chloramphenicol (Chloromycetin), tetracyclines, neomycin, and possibly other antibiotics may prolong prothrombin time in patients on oral anticoagulant drugs, mainly by interfering with vitamin K

production by gut bacteria. Quinine, quinidine, norethandrolone (Nilevar), and dextrothyroxine (Choloxin) also increases the anticoagulant effects of coumarin, though the mechanisms are unclear.

Coumarins elevate serum concentrations of diphenylhydantoin, probably by inhibiting enzymatic degradation of diphenylhydantoin in the liver. Diphenylhydantoin toxicity has been reported in patients receiving both drugs. Coumarins also potentiate the hypoglycemic effect of tolbutamide (Orinase) apparently by inhibiting its conversion to carboxytolbutamide in the liver.

Recently, clofibrate (Atromid S) has been found to increase anticoagulant activity when it is added to a regimen already containing coumarin derivatives. We, therefore, suggest that when clofibrate is added to the regimen under such conditions, the coumarin should be reduced to approximately one third to one half of its former dosage, and the prothrombin time checked frequently. The coumarin may then be gradually increased toward its former dosage. It will usually require a lower dosage than before the administration of clofibrate to produce optimal therapeutic response. The actions that are described above are reversed when these drugs are discontinued and it is essential to readjust the anticoagulant dosage accordingly.

Hospital Orders

The manner in which the orders are written is of importance. It is advisable to require on or near the front of each hospital chart an anticoagulant sheet which specifically requires the daily entry of the prothrombin time of the patient, the control prothrombin time for that day, and the dosage of the drug administered. It is thereby easily possible to note the trends of the response and take appropriate steps before extreme situations arise. If there is a prolongation of the prothrombin time above the optimal therapeutic level, it is wise to decrease the dose of the anticoagulant. On the other hand, if it seems to be shortening significantly, the indication for increasing the dose is clear.

Additional Tests for Control

Various tests are used in addition to the Quick one stage test and its modifications, and a wide variety of thromboplastins are available. These thromboplastins vary greatly in strength and action without adding significantly to the effectiveness or safety of the therapy. The loyalty of various workers to the tests they have developed or used has proved to be a major handicap in the standardization of anticoagulant therapy on a national or international basis. The International Committee on Thrombosis and Hemostasis is endeavoring to select a thromboplastin which will be adopted as an international standard.

One example of the problem is presented by the Thrombotest (Owren of Norway) which requires that a lower level of activity (8 to 10 percent) be adopted as the optimal therapeutic level of activity as compared with 20 to 25 percent. Lack of understanding of this fact has confused clinicians and invalidated some large scale studies.

When the Thrombotest indicates a level below five percent, it does not indicate how far below five percent it may be. Thus it fails to indicate how much vitamin K_1 one should give as an antidote. Otherwise, we have found it to be satisfactory. It may be used both as a finger puncture test and a vein test which is advantageous.

Whatever thromboplastin is used, whether it be Permaplastin, Simplastin, Acuplastin, or a local brain thromboplastin product, it is essential that the physician clearly understand the action of that particular thromboplastin in terms of therapy for his patient. He must become acquainted with the values of the prothrombin times or percentage activity which are optimal and not dangerous as reported in his own hospital or geographical area. This requires evaluation of the performance of the laboratory responsible for the data.

Antidotes

For an antidote to excessive oral anticoagulant use vitamin K_1. This is usually administered in dosages of 5 mg or less. Oral administration is usually satisfactory since it acts within three to six hours, almost as rapidly as intravenous administration. It is rarely necessary to use larger dosages. If the patient is hemorrhaging briskly, 10 mg may be given followed by an additional 5 mg in a few hours if the prothombin time does not return toward normal. When the therapy is controlled by physicians who are experienced with its use, serious hemorrhages are very rare. The causes of unpredictable hemorrhage include: unrecognized ulcers, unrecognized cancer of the gastrointestinal, urological, or respiratory tracts, or the result of an accident or injury. There have been numerous uses in which this has led to the discovery of a previously undiagnosed lesion.

Whole blood transfusions may be given for massive hemorrhage, but at the New York Hospital these have rarely been necessary in recent years.

When the prothrombin time reaches a level of 45 or 50 seconds against a control of 12 to 15 seconds, even in the absence of bleeding, it is advisable to give 5 mg of vitamin K_1 and to omit a dose or two of the anticoagulant. This in a sense acts as a buffer, preventing the prothrombin time from becoming excessive, and thereby decreasing the risk of bleeding.

Heparin rarely requires an antidote because its action is fairly short-lived, but if necessary, protamine sulfate, milligram for milligram, may be used. This is rarely necessary.

Hospital Protocol

It is essential that these patients be observed daily. The blood sample should be taken each morning and the prothrombin time reported by noon time. This should be checked by the physician or nurse prior to the administration of the daily dose at approximately 6 p.m.

An example of a useful schedule is as follows:

First day, Coumadin (warfarin sodium) 25 mg; second day, 15 mg; third day, 5 mg; thereafter, 5 mg daily, unless the prothrombin time exceeds 25 seconds (with a control time of 12 to 14). This provides an automatic cut-off. If the prothrombin time exceeds 25 seconds, the nurse does not administer the daily dose pending review by the physician. Extensive experience has shown this to be a safe procedure. It has been proven that more errors can occur if the dosage is ordered each day. The attending physicians or even the resident may fail to check the prothrombin time occasionally, and a dose may be omitted. This is a common error in many hospitals. If the prothrombin time is less than one and one-half times the control time, the dosage should be increased appropriately.

The Ambulatory Patient

In preparation for discharge the patient must be carefully instructed regarding the dose of anticoagulant he is to take each day until he is to see his physician. This must be written out or provided in chart form so that there is no chance of error. It has been found that it is better to use a standard dosage strength for a given drug. For example, in the use of warfarin sodium, we only use 5 mg tablets. The use of 2.5 mg, 7.5 mg, and 10 mg tablets tends to confuse the patient as to which tablet he has taken, and in telephone conversations he may make an error in trying to tell his physician. If a 5 mg tablet is used he may be instructed to cut it in half, or take two tablets. Both the patient and the physician should know precisely the dose taken. After discharge the patient should have a prothrombin time test within three or four days, then once a week if he seems to be well controlled. This may continue on a weekly basis for several weeks, and ultimately perhaps extend to two or even three weeks. Longer intervals between tests are not recommended; although the drug remains standardized, the condition of the patient may not. Infections, diarrhea, alcoholic binges, lack of food, and as previously discussed, various medications may influence the prothrombin time. Each patient should carry a card stating that he is on anticoagulant therapy and the name, address, and telephone number of his physician. Suitable cards are available from the American Heart Association.

Duration of Treatment

The question as to how long patients should be treated depends upon the condition under treatment. A patient with a simple thrombophlebitis, which subsides readily, should be on anticoagulants for a month or six weeks. Short periods of treatment (a week or less) are frequently followed by a relapse, sometimes with serious pulmonary emboli. On the other hand, the patients who have recurrent thromboembolism, such as pulmonary emboli, transient ischemic attacks, or myocardial infarctions, may be carried for many months or even years. There are now patients who have been on anticoagulant therapy for more than twenty years without any evidence of liver, renal, or other complications as a result of their anticoagulant therapy. The risk of serious hemorrhage must be weighed against the risk of the thromboembolic disease of the patient. Inadequate therapy cannot be expected to produce satisfactory results.

(The references may be seen in the original article.)

ACUTE ABDOMINAL PAIN IN CHILDHOOD, WITH SPECIAL REFERENCE TO CASES NOT DUE TO ACUTE APPENDICITIS*

Peter F. Jones,† M CHIR FRCS, Reprinted from the British Medical Journal, 1969, 1, 284–286, by permission of the Author and Editor.

Summary: Appendicitis is not the only common cause of acute abdominal pain in childhood. Almost equally common is an acute episode which in its early stages resembles acute appendicitis but which subsides without treatment in 24 to 48 hours. The clinical features of this syndrome are contrasted with those of appendicitis. The two conditions cannot always be distinguished on clinical grounds, leading to admission to hospital for observation and the finding of a normal appendix in 14% of operations for suspected appendicitis. Reasons are given for abandoning attempts to diagnose acute mesenteric adenitis at the bedside.

Introduction

Acute appendicitis in childhood rightly receives much attention because it is a potentially lethal condition. It is, however, not the only common cause of acute abdominal pain in childhood, and the purpose of this paper is to examine this wider subject.

The Royal Aberdeen Hospital for Children admits virtually all paediatric abdominal emergencies occurring in a population of some 400,000 people. Whenever the family doctor requests an emergency admission the child is accepted and is admitted direct to the ward. A previous paper (Winsey and Jones, 1967) described a detailed survey of every case of acute abdominal pain admitted to this hospital during 1965. It showed that of cases admitted to surgical wards roughly half were treated surgically, 37% having acute appendicitis, 7% some other major surgical conditions such as intussusception, and in 6% a normal appendix was removed. In the nonsurgical group 15% had an acute medical disease such as pneumonia or pyelitis, but 35% showed a steady spontaneous improvement of their illness after admission and left hospital in 48 to 72 hours.

It seemed surprising that so many children admitted with a suspected acute surgical abdominal lesion should recover spontaneously and quickly, therefore it was decided to continue the survey for a further two years, 1966 and 1967. The method of recording previously used was continued.

* Based on a paper read to the Annual Meeting of the British Association of Paediatric Surgeons, Liverpool, July 1968.

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Findings

During the three years 785 children up to 12 years of age were admitted to the hospital as suspected cases of acute appendicitis and treated without mortality. The final outcome in these 785 children is set out in Table I.

TABLE I. *Outcome of Admissions to Surgical Wards for Suspected Appendicitis, Children's Hospital, Aberdeen, 1965–7*

Outcome of Admission	1965	1966	1967	Total	
Appendicectomies:					
Acute appendicitis found	114	121	163	398	
Normal appendix found:					
Mesenteric adenitis	11	11	20	42	14% of laparotomies
Others	10	9	3	22	
No operation:					
Spontaneous recovery	119	100	104	323	
Total	254	241	290	785	

It will be seen that the numbers in each group in each year are comparable, though there is no known factor responsible for the appreciable increase in acute appendicitis during 1967.

In particular it will be noted that about 40% of those admitted with suspected acute appendicitis settled without specific treatment. When sent into hospital these children all had symptoms and/or signs which worried their family doctor (and in some cases a surgeon called to the home on a domiciliary consultation). Analysis of age shows few patients below 5 years (16%) and an even incidence throughout the seven school years (with a slight preponderance at 9 years of age). Boys (55%) were slightly more affected than girls (45%), and this was seen at all ages.

A study of the clinical picture in the 323 children who settled spontaneously confirms the description of this group given by Winsey and Jones (1967). Pain is usually felt centrally (70%), much less often in the right iliac fossa (13%). Pain only rarely shifts to the right iliac fossa, and the patient can move without aggravating the pain. Tenderness is usually present in the right iliac fossa initially (this is the main reason for the high admission rate), but it soon relents, and is found in only half the patients

by the time they are admitted. Rebound tenderness is not elicited and muscle guarding is most unusual.

When set out in this way this condition is clearly distinguishable from acute appendicitis (Table II), but these points are not always so clear-cut in the individual case, more especially in the early hours of the illness. It is the steady amelioration of symptoms and signs seen when the patient is re-examined at intervals of a few hours which enables this syndrome of acute non-specific abdominal pain of childhood to be identified: it is a diagnosis which can firmly be made only in retrospect.

TABLE II. *Comparison of Incidence of Symptoms and Signs of Most Assistance in Distinguishing Acute Appendicitis from Acute Nonspecific Abdominal Pain of Childhood*

Symptom or Sign	Acute Appendicitis	Abdominal Pain
Shift of pain	64%	14%
Worse on movement:		
Symptom	75%	25%
Sign	60%	6%
Tender in right iliac fossa	100%	55%
Guarding	90%	8%
Rebound	80%	1%

It seemed wise to look further into the subsequent history of all these children, to see whether any later event might shed light on the reason for the original admission. Seven out of the 323 children were readmitted up to June 1968 with surgical abdominal disease—six had acute appendicitis and one had an intussusception of Meckel's diverticulum. With the benefit of hindsight it is possible to say that three of the six children probably had acute appendicitis on their first admission but in each case it was at a stage when it was clinically improving. Another nine children were readmitted with abdominal pain which settled quickly, one child was readmitted with a urinary infection (which was not present on the first admission) and another was admitted with torsion of a normally sited left testicle. There were no other relevant readmissions.

The other main group comprised 462 children who had an appendicectomy because acute appendicitis was either confidently diagnosed or strongly suspected on the basis of one or more examinations after admission.

In 398 children an acutely inflamed appendix was removed, the diagnosis being confirmed by naked-eye and histological examination. The clinical features of this group were described in detail in the previous paper.

In 64 children the appendix was normal when exposed at operation. In 42 of these children acute mesenteric adenitis was found. In six cases various other conditions, such as haemorrhage into an ovarian cyst, threadworms in the appendix, or suppurating mesenteric nodes, were thought to be the cause of the illness. In the remaining 16 children laparotomy did not yield any recognizable cause for their pain.

Acute mesenteric adenitis caused considerable confusion. The histories of these 42 patients were examined in an attempt to elucidate this. In one-quarter of them there was a shift of pain from the centre of the abdomen to the right iliac fossa, one-third found it painful to move about the bed, and another one-third had rebound tenderness. Half the patients had guarding as well as tenderness in the right iliac fossa, and in nearly half these signs became more pronounced while these subjects were observed in the ward. These were features characteristic of acute appendicitis, and it was clearly necessary to open these patients. Nearly half of them had the central colicky abdominal pain which Aird (1945) considered to be typical of acute mesenteric adenitis, but this in itself did not allow physical signs strongly suggestive of appendicitis to be overlooked. In only 4 of the 42 patients were cervical lymph nodes palpably enlarged.

Discussion

The analysis of a large group of children admitted to hospital with acute abdominal pain shows that in the first hours of the illness it can be difficult to distinguish those who have acute appendicitis from those who have transient non-specific abdominal pain. Acute abdominal pain and tenderness in the right iliac fossa are seen in both conditions. The difficulties of diagnosing atypical acute appendicitis are widely known and as a consequence considerable numbers of children with non-specific abdominal pain are referred to hospital—roughly 20,000 every year in Great Britain, if experience in Aberdeen is representative of practice throughout the country.

The two groups can to a large extent be separated by the practice of active observation, in which clinical examination is repeated every few hours until a diagnosis is reached. The more serious cases of appendicitis are evident on the first examination and their treatment is not delayed by this practice. In the other cases no harm comes from observation for a few hours, and often a very much more satisfactory examination is possible on a second visit. Repetition

of the examination may be possible in the child's home, but circumstances or distance may make this unwise and the child is then referred to hospital at the first visit.

In Aberdeen some 40% of children referred to hospital with suspected acute appendicitis are found to improve spontaneously within hours or at most a day or so. The pathological condition which causes the acute and definite pain and tenderness these children show remains obscure. Constipation is often incriminated, but it was not noticeable in more than a small minority (8%). Serious consideration must be given to the possibility that these children have mild appendicitis. However, only six were readmitted with acute appendicitis over three and a half years, and the policy throughout was to advise operation whenever doubt regarding the diagnosis persisted. It seems unlikely that many acutely inflamed appendices were not removed.

Some of the children may have had acute mesenteric adenitis, but there can be no certainty about this. Our experience with this disease must cast great doubt on whether it is wise or safe to attempt to diagnose it at the bedside. We fully endorse the conclusion of Potts (1959), who stated: "Acute mesenteric lymphadenitis so closely mimics acute appendicitis that differentiation is impossible. If you make a diagnosis of acute mesenteric lymphadenitis it is wise to advise appendectomy to see whether you are right."

Even with the experience and investigative resources available in hospital, and the opportunity to re-examine at intervals, it is not possible to make a certain division between those children needing and those not needing an operation. Decision has to be based on continuing clinical assessment, and in this series 14% of the children thought to need operation were found to have a normal appendix.

Information about the number of normal appendices removed in operations for suspected acute appendicitis is not always given, but some papers in which this information is provided are summarized in Table III. The figure of 14% for the proportion of normal appendices seems to be the commonest and the lowest figure quoted. It is also associated with the least mortality, so it does not appear that a greater readiness to explore the doubtful case will necessarily lower mortality.

Howie (1968a) suggested that a radical approach, with removal of the appendix in four out of every five patients admitted with suspected appendicitis, will lead to more acutely inflamed appendices being removed and will produce lowering of morbidity after discharge. The approach in this hospital has been radical in so far as operation has always been performed whenever doubt persisted about the existence of acute appendicitis. This has led to three appendicectomies being done among every five patients admitted—a rate which Howie considers to indicate a "conservative approach." However, this series was composed entirely of children, while Howie studied no patients under 12 years. It may well be that acute non-specific abdominal pain in childhood is a different condition, does not imply there is any appendicular infection, and can generally be identified by its tendency toward rapid spontaneous resolution. Certainly the readmission rate among our young patients is very low—about 4%—and we have not experienced any indication for the planned appendicectomy for recurrent abdominal pain which Howie (1968b) described among his patients who were treated conservatively.

TABLE III.—*Incidence of Finding of Normal Appendix at Operation for Suspected Acute Appendicitis*

Authors	City	Age Range in Years	Total Appendicectomies	Normal Appendices Removed		Mortality (%)
				No.	%	
Boles *et al.* (1959)	Columbus	Up to 12	977	140	14	0–1
Brown (1956)	Edinburgh	Up to 12	176	25	14	0–14
Present series	Aberdeen	Up to 12	462	64	14	0
McLauthlin and Packard (1961)	Denver	Up to 14	474	107	22	0–86
Barnes *et al.* (1962)	Boston	Adults	7,820	1,978	25	1–0

However, nothing should detract from the support which Howie's work gives to the policy of operation when doubt persists about the presence of acute appendicitis; this must be strongly endorsed.

It may at first sight seem to be unsatisfactory that 14 out of every 100 children operated on for suspected appendicitis will have a normal appendix and that two out of every five children admitted with this diagnosis will recover without treatment. These figures express, however, a widespread concern about the difficulty of diagnosing some cases of acute appendicitis in childhood. If the mortality of acute appendicitis is now very low, it must not be forgotten that a number of children still reach hospital gravely ill

with peritonitis and survive only after much discomfort, hard work, and anxiety.

Readiness to admit and to operate on the doubtful case is the price we rightly pay for the earlier solution of the diagnostic problems which acute appendicitis in childhood will continue to set.

(The references may be seen in the original article.)

DRUGS FOR PARASITIC INFECTIONS

The Medical Letter 11(6) Issue 266:21–28, March 21, 1969.

(*Note*: This special issue of The Medical Letter brings up to date the tables in the November 8, 1963 issue on drugs for parasitic infections. This issue also includes a new table of adverse effects of antiparasitic drugs. The tables are based on the clinical experience of Medical Letter consultants, including those in tropical areas, on the recommendations of the World Health Organization and of the manufacturers of the drugs, and on the medical literature. The tables and comments reflect a consensus, not unanimity. Changes and additions, as needed, will be reported in the quarterly index issues of The Medical Letter.)

Parasitic infections are by no means limited to tropical and subtropical countries; they also occur in other areas, especially where living standards are low. With increasing world travel, physicians anywhere may encounter infections caused by parasitic organisms. This issue, like that for microbial infections (Medical Letter, Vol. 10, p. 77, Oct. 4, 1968), is intended to help the physician select the drugs offering the best combination of effectiveness and relative safety; alternative drugs for use against each parasite are also listed. The table starting on page 23 lists the major adverse effects of the drugs.

Dosages—Because most of the drugs can have serious adverse effects, dosage recommendations are also given and should be exceeded only with special caution. Dosage for infants and children is on a less secure basis than for adults; when doses are not given in milligrams per kilogram of body weight, they should take into account possibly greater toxicity in infants and young children.

Adverse Effects—Adverse effects are most likely to occur in patients who are poorly nourished or who also have other infections. When the first-choice drug is ineffective and the alternative drug is seriously hazardous, it may be desirable to try a second course of treatment with the first-choice drug before using the alternative drug. The possibility of unexpected interactions with other drugs administered concurrently should be kept in mind. In patients with liver disease, some of the drugs may not be well metabolized, with consequent cumulative effects and greater toxicity. Drugs excreted mainly by the kidneys are especially toxic in patients with impaired renal function. Gastric irritation caused by some drugs may be reduced if they are taken with meals. In severe disease, consultation with a specialist in parasitic infections is desirable.

TABLE OF DRUGS FOR PARASITIC INFECTIONS

(Most of the drugs listed in the following table can have serious adverse effects. It is important therefore that the table be used in conjunction with the table of adverse effects which begins on page 23. The manufacturers' package inserts should also be consulted. As the footnotes indicate, some of the drugs are not readily available in the United States.)

Infecting Organism	*Drug of Choice*	*Adult Dose*	*Alternative Drugs*	*Adult Dose*
Roundworms, Nematodes				
Ascaris lumbricoides (roundworm)[1]	piperazine citrate	75 mg/kg (maximum 3.5 Gm) daily for 2 days	thiabendazole	25 mg/kg b.i.d. for 2 days
Trichuris trichiura (whipworm)[2]	thiabendazole [2]	25 mg/kg b.i.d. for 2 days	hexylresorcinol [3] (0.1% solution)	500 ml by rectal retention for 1 hour

Infecting Organism	*Drug of Choice*	*Adult Dose*	*Alternative Drugs*	*Adult Dose*
Necator americanus (hookworm)[4]	tetrachloroethylene [5]	a single dose of 0.12 ml/kg (maximum 5 ml)	thiabendazole	25 mg/kg b.i.d. for 2 days
	or			
	bephenium	one 5-Gm packet b.i.d. for 3 days		
Ancylostoma duodenale (hookworm)[4]	bephenium	one 5-Gm packet b.i.d. for 1 day	tetrachloroethylene [5]	a single dose of 0.12 ml/kg (maximum 5 ml)
			thiabendazole	25 mg/kg b.i.d. for 2 days
Strongyloides stercoralis	thiabendazole	25 mg/kg b.i.d. for 2 days	pyrvinium pamoate	single dose, 5 mg/kg (maximum 250 mg); repeat after 2 weeks
Enterobius (Oxyuris) vermicularis (pinworm)	piperazine citrate	65 mg/kg (maximum 2.5 Gm) daily for 8 days	thiabendazole	25 mg/kg b.i.d. for 1 day; repeat in 7 days
	or			
	pyrvinium pamoate	single dose, 5 mg/kg (maximum 250 mg); repeat after 2 weeks		
Trichinella spiralis (trichinosis)	no specific therapy; corticosteroids for severe symptoms	20 to 40 mg (prednisone) daily, reduced after 3 to 5 days	thiabendazole [6]	25 mg/kg b.i.d. until symptoms subside or toxic effects occur
Cutaneous larva migrans (creeping eruption) (dog and cat hookworm in man)	thiabendazole	25 mg/kg b.i.d. for 2 days, repeat in 2 days if necessary; or apply as topical ointment	none	
Visceral larva migrans [7] (dog and cat roundworm in man)	no specific therapy; corticosteroids for severe symptoms	20 to 40 mg (prednisone)daily, reduced after 3 to 5 days	thiabendazole [8]	25 mg/kg b.i.d. until symptoms subside or toxic effects occur
			diethylcarbamazine [8]	120 mg t.i.d. for 30 days
Filaria				
Wuchereria bancrofti W. (or Brugia) malayi Loa loa (tropical eosinophilia)	diethylcarbamazine [9]	2 mg/kg t.i.d. for about 14 days [10]	none	
Onchocerca volvulus (river blindness)	diethylcarbamazine [9]	25 mg daily for 3 days 50 mg daily for 3 days 100 mg daily for 3 days 150 mg daily for 12 days	none	
	plus			
	suramin [11]	100 to 200 mg (test dose) i.v., then 1 Gm i.v. at weekly intervals for 5 weeks		
Dracunculus medinensis (guinea worm)	niridazole [12]	25 mg/kg daily for 7 days	none	

Infecting Organism	*Drug of Choice*	*Adult Dose*	*Alternative Drugs*	*Adult Dose*
Tapeworms, Cestodes				
Taenia saginata (beef tapeworm) Diphyllobothrium latum (fish tapeworm)	niclosamide [11]	4 tablets (2 Gm) chewed thoroughly in a single dose after a light meal	aspidium oleoresin [12]	4 to 8 Gm plus 8 Gm acacia in water; one-half of dose is taken early in morning, the rest one hour later; or single dose of aspidium by duodenal tube to prevent vomiting
	or quinacrine hydrochloride	4 doses of 200 mg 10 minutes apart; 600 mg sodium bicarbonate with each dose		
Taenia solium (pork tapeworm)	quinacrine hydrochloride	4 doses of 200 mg 10 minutes apart; 600 mg sodium bicarbonate with each dose	aspidium oleoresin [12]	4 to 8 Gm plus 8 Gm acacia in water; one-half of dose is taken early in morning, the rest one hour later; or single dose of aspidium by duodenal tube to prevent vomiting
—larval stage (cysticercosis)	none			
Hymenolepis nana (dwarf tapeworm)	niclosamide [11]	4 tablets (2 Gm) chewed thoroughly in a single dose each day for 5 to 7 days	quinacrine hydrochloride	4 doses of 200 mg 10 minutes apart; 600 mg sodium bicarbonate with each dose; repeat in 1 or 2 weeks if necessary
Echinococcus granulosus (larval stage) (echinococcosis) (dog and wolf tapeworm in man)	none			
Flukes, Trematodes				
Schistosoma haematobium	niridazole [12]	25 mg/kg daily for 5 to 7 days	lucanthone [12] (only for children under 16 because of toxicity in adults)	15 mg/kg daily in 3 doses for 6 to 8 days
	or stibophen [13] (8.5 mg antimony per ml)	4 ml daily i.m. 5 days per week to a total of 80 ml	antimony sodium dimercaptosuccinate [11, 13]	total dosage of 40 mg/kg in 5 divided doses given i.m. once or twice a week
Schistosoma mansoni	stibophen [13] (8.5 mg antimony per ml)	4 ml daily i.m. 5 days per week to a total of 80 to 100 ml	lucanthone [12] (only for children under 16 because of toxicity in adults)	15 mg/kg daily in 3 doses for 6 to 8 days
	or antimony sodium dimercaptosuccinate [11, 13]	total dosage of 40 mg/kg in 5 divided doses given i.m. once or twice a week	niridazole [12]	25 mg/kg daily for 7 days

Infecting Organism	*Drug of Choice*	*Adult Dose*	*Alternative Drugs*	*Adult Dose*
Schistosoma japonicum	antimony potassium tartrate [14] (0.5% solution)	i.v. doses of 8, 12, 16, 20, 24, and 28 ml on alternate days, continuing at 28 ml on alternate days for 10 doses	stibophen [13] (8.5 mg antimony per ml)	8 to 10 ml per day (a very toxic amount) i.m. for 10 days or longer as required
			antimony sodium dimercaptosuccinate [11, 13]	total dosage of 40 mg/kg in 5 divided doses given i.m. once or twice a week
Clonorchis sinensis (liver fluke)	chloroquine phosphate [15]	250 mg t.i.d. for 6 weeks	bithionol [8, 11]	30 to 50 mg/kg on alternate days for 10 to 15 doses
Paragonimus westermani (lung fluke)	bithionol [11]	30 to 50 mg/kg on alternate days for 10 to 15 doses	chloroquine phosphate [15]	250 mg t.i.d. for 6 weeks
Fasciola hepatica (sheep liver fluke)	emetine hydrochloride [16]	20 to 65 mg daily i.m. for 8 to 10 days	bithionol [8, 11]	30 to 50 mg/kg on alternate days for 10 to 15 doses
	or			
	dehydroemetine dihydrochloride [11, 16]	1 mg/kg daily i.m. for 10 days	chloroquine phosphate [8, 15]	250 mg t.i.d. for 6 weeks
Protozoa				
Entamoeba histolytica				
—mild or asymptomatic intestinal infection	diiodohydroxyquin	650 mg t.i.d. for 20 days	none	
—severe intestinal disease	emetine hydrochloride [16]	1 mg/kg (maximum 65 mg) daily, subcutaneously or i.m. for 10 days	metronidazole [8]	800 mg t.i.d. for 10 days (three times the dose recommended for other conditions)
	or			
	dehydroemetine dihydrochloride [11, 16]	1 to 1.5 mg/kg daily i.m. or subcutaneously for 10 days (maximum total dose 1 Gm)		
	plus			
	tetracycline (with either emetine or dehydroemetine)	250 mg q.i.d. for 5 days		
	followed by			
	diiodohydroxyquin	650 mg t.i.d. for 20 days		
—hepatic abscess	emetine hydrochloride [16]	1 mg/kg (maximum 65 mg) daily, subcutaneously or i.m. for 7 to 10 days	metronidazole [8]	800 mg t.i.d. for 10 days (three times the dose recommended for other conditions)
	or			
	dehydroemetine dihydrochloride [11, 16]	1 to 1.5 mg/kg daily i.m. or subcutaneously for 10 days (maximum total dose 1 Gm)		
	plus			
	chloroquine phosphate (with either emetine or dehydroemetine)	1 Gm daily for 2 days, then 500 mg daily for 2 to 3 weeks		

Infecting Organism	*Drug of Choice*	*Adult Dose*	*Alternative Drugs*	*Adult Dose*
Dientamoeba fragilis	diiodohydroxyquin [8]	650 mg t.i.d. for 10 days	a tetracycline	250 mg q.i.d. for 7 days
Giardia lamblia	quinacrine hydrochloride	100 mg t.i.d. for 5 to 7 days	metronidazole [8]	250 mg t.i.d. for 10 days
Trichomonas vaginalis	metronidazole	250 mg t.i.d. for 10 days	topical agents [17]	
Balantidium coli	oxytetracycline	500 mg q.i.d. for 10 days	diiodohydroxyquin [8]	650 mg t.i.d. for 20 days
Pneumocystis carinii	pentamidine isethionate [11]	4 mg/kg i.m. daily for 12 to 14 days	pyrimethamine [8, 18]	25 mg daily in divided doses for 12 to 14 days
			plus sulfadiazine	2 Gm daily in divided doses for 12 to 14 days
Various Blood and Tissue Parasites				
Plasmodia (malaria)				
—suppression of disease while in endemic area and prevention of attack (P. vivax, P. ovale, and P. malariae) after departure (prophylaxis)	chloroquine phosphate [19]	500 mg (300 mg base) once weekly and continued for 6 weeks after last exposure in endemic area	amodiaquine dihydrochloride	520 mg (400 mg base) once weekly and continued for 6 weeks after last exposure in endemic area
	plus primaquine phosphate [20, 21]	26.3 mg (15 mg base) daily for 14 days after last exposure in endemic area	*plus* primaquine phosphate [20, 21]	26.3 mg (15 mg base) daily for 14 days after last exposure in endemic area
—treatment of uncomplicated attack (all plasmodia except resistant P. falciparum)	chloroquine phosphate [19, 22]	1 Gm (600 mg base), then 0.5 Gm in 6 hours, then 0.5 Gm daily for 2 days	amodiaquine dihydrochloride	780 mg (600 mg base) first day, then 520 mg (400 mg base) daily for 2 days
—treatment of severe illness, parenteral dosage (all plasmodia except resistant P. falciparum)	chloroquine hydrochloride [19]	250 mg (200 mg base) i.m. every 6 hours (maximum 1 Gm daily) until oral therapy is possible	none	
—prevention of relapses ("radical" cure after "clinical" cure) (all plasmodia except P. falciparum)	primaquine phosphate [21]	26.3 mg (15 mg base) daily for 14 days	none	
P. falciparum (resistant strains)[22, 23]				
—treatment of uncomplicated attack	quinine sulfate [24]	650 mg t.i.d. for 14 days	sulformethoxine [12, 25]	a single dose of 1 Gm
	plus pyrimethamine [18]	25 mg b.i.d. for 3 days	*plus* pyrimethamine [18]	a single dose of 50 mg
	plus either sulfadiazine	500 mg q.i.d. for 5 days		
	or dapsone	25 mg daily for 28 days		

Infecting Organism	*Drug of Choice*	*Adult Dose*	*Alternative Drugs*	*Adult Dose*
—treatment of severe illness, parenteral dosage	quinine dihydrochloride [26]	600 mg in 300 ml normal saline i.v. over at least 30 minutes; repeat in 6 to 8 hours until oral therapy is possible	none	
Leishmania				
L. donovani (kala azar, visceral leishmaniasis)	antimony sodium gluconate [11, 27]	600 mg i.m. or i.v. daily for 6 to 10 days; course may be repeated for resistant cases	pentamidine isethionate [11, 27]	2 to 4 mg/kg i.m. daily for up to 15 doses
L. tropica (oriental sore, cutaneous leishmaniasis)	antimony sodium gluconate [11]	600 mg i.m. or i.v. daily for 6 to 10 days	topical agents [28]	
L. braziliensis (American mucocutaneous leishmaniasis)	antimony sodium gluconate [11]	not certain, probably same as for other Leishmania infections	amphotericin B	0.25 to 1 mg/kg by slow infusion daily or every 2 days up to 8 weeks
			cycloguanil pamoate [8, 12]	a single i.m. injection of 350 mg base
Trypanosomes				
T. cruzi (South American trypanosomiasis, Chagas' disease)	none [29]			
T. gambiense (African trypanosomiasis, sleeping sickness)				
—hemolymphatic stage	pentamidine isethionate [11]	4 mg/kg i.m. daily for 10 days	suramin [11]	100 to 200 mg (test dose) i.v., then 1 Gm i.v. on days 1, 3, 7, 14, and 21
T. rhodesiense (African trypanosomiasis, sleeping sickness)				
—hemolymphatic stage	suramin [11]	100 to 200 mg (test dose) i.v., then 1 Gm i.v. on days 1, 3, 7, 14, and 21	pentamidine isethionate [11]	4 mg/kg i.m. daily for 10 days
T. gambiense or T. rhodesiense				
—late disease with CNS involvement	melarsoprol [11, 30]	2 to 3.6 mg/kg i.v. daily for 3 doses; after one week 3.6 mg/kg i.v. daily for 3 doses; may be repeated again after 10 to 21 days	tryparsamide [12]	one injection of 30 mg/kg i.v. every 5 days to a total of 12 injections; may be repeated after one month
			plus suramin [11]	one injection of 10 mg/kg i.v. every 5 days to a total of 12 injections; may be repeated after one month
—chemoprophylaxis [31]	pentamidine isethionate [11]	4 mg/kg i.m. every 3 to 6 months	suramin [11]	0.3 to 0.7 Gm i.v. every 2 to 3 months

Infecting Organism	*Drug of Choice*	*Adult Dose*	*Alternative Drugs*	*Adult Dose*
Toxoplasma gondii (toxoplasmosis)	pyrimethamine [18, 32]	25 to 50 mg daily for 1 month	none	
	plus			
	trisulfapyrimidines	2 to 6 Gm daily for 1 month		

[1] When there is infection by two or more species of intestinal helminths, Ascaris should always be treated first.

[2] Asymptomatic infections are best left untreated (except in food handlers). Thiabendazole cures only about 20 percent of cases. An experimental drug, dichlorvos, is reported to be more effective.

[3] Hexylresorcinol enemas should be given with care and only in a hospital. The drug is contraindicated in severe colonic disorders.

[4] Light hookworm infections need not be treated in the absence of symptoms or anemia; the mode of entry (through the skin) should be pointed out to the patients so that they can prevent additional infection.

[5] Tetrachloroethylene is usually available only as a veterinary drug; however, it has frequently been effectively and safely used for humans. It should always be given on an empty stomach. Because it can cause nausea and dizziness and, rarely, loss of consciousness, the patient should be kept at rest for four hours after the drug is administered.

[6] The efficacy of thiabendazole for trichinosis is not clearly established. It appears to allay symptoms and reduce eosinophilia but its effect on larvae which have migrated to muscle is questionable.

[7] Visceral larva migrans is usually a self-limiting disease. Treatment should be restricted to severe cases.

[8] Usefulness in this condition is still under investigation.

[9] Diethylcarbamazine should be administered with special caution in Loa loa because it can provoke an encephalopathy. Antihistamines or corticosteroids may be required to reduce allergic reactions due to disintegration of microfilaria in the treatment of all filarial infections, especially those caused by Onchocerca.

[10] Diethylcarbamazine may occasionally have to be given for as long as 30 days.

[11] In the United States, this drug is available from the Parasitic Disease Drug Service, Parasitic Diseases Branch, Epidemiology Program, National Communicable Disease Center, Atlanta, Georgia 30333 (March 1969).

[12] Not available for clinical use in the United States (March 1969).

[13] Stibophen and antimony sodium dimercaptosuccinate are contraindicated in renal and cardiac disease, and in hepatic disease not caused by schistosomiasis (but note that Schistosoma infections usually cause hepatic disease). Either drug should be stopped in the event of recurrent vomiting, progressive albuminuria, persistent joint pain, rash, or intercurrent infection.

[14] Antimony potassium tartrate should be given slowly, intravenously; if given rapidly it may produce a hacking cough, vomiting, and severe or fatal reactions.

[15] Chloroquine usually does not cure, but produces temporary suppression of ova.

[16] Because of the toxic effects of the drug on the heart, patients receiving emetine should remain sedentary during therapy. Dehydroemetine may prove to be as effective and possibly less toxic than emetine (see footnote 11).

[17] Metronidazole, a systemic agent, is effective in both females and males. Topical agents, such as arsenicals and hydroquinones, may be effective in females with mild infection.

[18] To prevent hematologic toxicity from pyrimethamine, it is advisable to administer folinic acid or calcium leucovorin, about 10 mg/day intramuscularly.

[19] Dosage is oral unless otherwise stated. The World Health Organization has suggested (1965) an initial dose of 10 mg chloroquine base per kg of body weight, and subsequent doses of 5 mg/kg.

[20] The routine use of primaquine for chemoprophylaxis in all civilians who have been in a malaria endemic area is questionable. Intensity of exposure should determine its use.

[21] Dosages of primaquine in excess of 26 mg (15 mg base) per day for 14 days may cause hemolytic anemia, especially in patients whose red cells are deficient in glucose-6-phosphate dehydrogenase. This deficiency is more common in Negroes but more severe in Caucasians.

[22] In falciparum malaria, if the patient has not shown a prompt response to conventional doses of chloroquine, parasite resistance to this drug must be considered.

[23] Chloroquine-resistant strains of P. falciparum have been reported from Brazil, Cambodia, Colombia, Guyana, Malaya, South Vietnam, and Thailand. At the present time (1969), most P. falciparum infections in American troops in Vietnam are considered to be resistant to chloroquine.

[24] Quinine alone will control an acute attack of resistant P. falciparum, but in a substantial proportion of infections, particularly strains from Southeast Asia, it fails to prevent recurrence. Addition of pyrimethamine, with either sulfadiazine or dapsone, substantially lowers the recurrence.

[25] A prolonged-action sulfonamide.

[26] Intravenous administration of quinine dihydrochloride can be hazardous, but it is preferred in very seriously ill patients. Constant monitoring of the pulse and blood pressure is necessary to detect arrhythmia or hypotension. Oral quinine sulfate should be substituted as soon as possible.

[27] Pentamidine should be used for failures with antimony and sometimes for initial treatment in cases from Sudan (which are often resistant to antimony). All solutions should be protected from light to avoid production of hepatotoxic compounds.

[28] In patients with few or single lesions that are not cosmetically significant, topical or local treatment (carbon dioxide snow and infrared and radiotherapy are among the agents that have been used) may be preferable to the risk of toxicity from systemic antimonial compounds.

[29] A nitrofurfurylidene derivative, Bayer 2502 (an investigational drug), is reported to be effective in many cases.

[30] In frail patients, begin with as little as 18 mg and increase the dose progressively. Pretreatment with suramin has been advocated for debilitated patients.

[31] For chemoprophylaxis of African trypanosomiasis (sleeping sickness), the risk of serious drug toxicity must be weighed against the risk of acquiring the disease. Chemoprophylaxis is usually not recommended for travelers on brief visits to endemic areas.

[32] In ocular toxoplasmosis, as much as 75 mg is recommended by some ophthalmologists; both drugs may have to be taken for more than a month. Corticosteroids may also be employed for anti-inflammatory effect on the eyes.

ADVERSE EFFECTS OF ANTIPARASITIC DRUGS

AMODIAQUINE DIHYDROCHLORIDE (Camoquin)

Occasional: vomiting; diarrhea; vertigo

Rare: corneal deposits; bluish-grey pigmentation of fingernails, skin and hard palate; retinopathy; polyneuropathy; liver damage

AMPHOTERICIN B USP (Fungizone)

Frequent: renal damage; hypokalemia; febrile reactions; thrombophlebitis at site of injection

Occasional: hypomagnesemia; normocytic, normochomic anemia

Rare: hemorrhagic gastroenteritis; blood dyscrasia; liver damage; maculopapular rash; blurred vision; peripheral neuropathy; convulsions; anaphylactic shock

ANTIMONY POTASSIUM TARTRATE USP

Frequent: coughing and vomiting when intravenous administration is rapid; muscle and joint stiffness; bradycardia

Occasional: colic, diarrhea; rash; pruritus; herpes zoster; renal damage; shock

Rare: hepatocellular damage; hemolytic anemia

ANTIMONY SODIUM DIMERCAPTOSUCCINATE (STIBOCAPTATE) (Astiban)

Adverse effects similar to effects of ANTIMONY POTASSIUM TARTRATE, but, except for rash and pruritus, less frequent and usually less severe

ANTIMONY SODIUM GLUCONATE, trivalent (Triostam)

Adverse effects similar to effects of ANTIMONY POTASSIUM TARTRATE, but less frequent and usually less severe

ANTIMONY SODIUM GLUCONATE, pentavalent (Pentostam)

Adverse effects similar to effects of ANTIMONY POTASSIUM TARTRATE, but less frequent and usually less severe

ASPIDIUM OLEORESIN

Occasional: vomiting; diarrhea; polyneuropathy

Rare: convulsions; coma; blindness; liver and kidney damage

BEPHENIUM HYDROXYNAPHTHOATE (Alcopara)

Occasional: vomiting; diarrhea

BITHIONOL

Frequent: photosensitivity skin reaction; vomiting; diarrhea, abdominal pain; urticaria

CHLOROQUINE HYDROCHLORIDE and *CHLOROQUINE PHOSPHATE USP* (Aralen)

Frequent: pruritus; vomiting; headache

Occasional: depigmentation of hair; skin eruptions; corneal opacity; irreversible retinal injury (especially when total dosage exceeds 100 Gm); weight loss; partial alopecia; extraocular muscle palsies; blood dyscrasia

Rare: discoloration of nails and mucous membranes of mouth; nerve-type deafness; photophobia

DAPSONE (Avlosulfon)

Frequent: rash

Occasional: blood dyscrasia, including hemolytic anemia; nephrotic syndrome; liver damage

DEHYDROEMETINE (see EMETINE HYDROCHLORIDE)

DIETHYLCARBAMAZINE CITRATE USP (Hetrazan)

Frequent: severe allergic or febrile reactions due to disintegration of worms

Occasional: encephalopathy

DIIODOHYDROXYQUIN USP (Diodoquin)

Occasional: rash; acne; slight enlargement of the thyroid gland; nausea; cramps; anal pruritus

EMETINE HYDROCHLORIDE USP

Frequent: cardiac arrhythmias; precordial pain; muscle weakness

Occasional: diarrhea; vomiting; peripheral neuropathy; heart failure

LUCANTHONE HYDROCHLORIDE USP

Frequent: vomiting; headache; dizziness; depression and anxiety; abdominal pain

Occasional: yellow discoloration of skin and sclera (*Note:* Lucanthone should not be used in adults because of the frequency of serious adverse effects.)

MELARSOPROL (Mel B)

Frequent: myocardial damage; albuminuria; hypertension; colic; Herxheimer-type reaction; encephalopathy; vomiting; peripheral neuropathy

Rare: shock

METRONIDAZOLE (Flagyl)

Frequent: nausea; headache

Occasional: vomiting; diarrhea; insomnia; weakness; stomatitis; vertigo; paresthesia; rash

Rare: ataxia

NICLOSAMIDE (Yomesan)

Occasional: nausea; abdominal pain

NIRIDAZOLE (Ambilhar)

Frequent: vomiting; diarrhea; cramps; dizziness; headache

Occasional: slight electrocardiographic changes; rash; insomnia; paresthesia

Rare: convulsions; psychosis; hemolytic anemia in G-6-PD-deficient persons

PENTAMIDINE ISETHIONATE (Lomidine)

Frequent: hypotension; vomiting; blood dyscrasia

Occasional: may aggravate diabetes; shock

Rare: Herxheimer-type reaction; renal injury

PIPERAZINE CITRATE USP (Antepar)

Occasional: dizziness; urticaria

Rare: exacerbation of epilepsy; visual disturbances

PRIMAQUINE PHOSPHATE USP (Primaquine)

Frequent: hemolytic anemia in G-6-PD-deficient persons

Occasional: neutropenia; GI disturbances; methemoglobinemia in G-6-PD-deficient persons

Rare: CNS symptoms; hypertension; arrhythmias

PYRIMETHAMINE USP (Daraprim)

Occasional: blood dyscrasia; folic acid deficiency

Rare: rash; vomiting; convulsions; shock

PYRVINIUM PAMOATE USP (Povan)

Frequent: turns stools red

Occasional: vomiting; diarrhea

Rare: photosensitivity skin reactions

QUINACRINE HYDROCHLORIDE USP (Atabrine)

Frequent: dizziness; headache; vomiting

Occasional: toxic psychosis; blood dyscrasia; urticaria; severe exfoliative dermatitis; yellow staining of skin and sclera; blue and black nail pigmentation; ocular effects similar to those caused by chloroquine

Rare: acute hepatic necrosis

QUININE DIHYDROCHLORIDE and *QUININE SULFATE*

Frequent: arrhythmias; hypotension; cinchonism (tinnitus, headache, nausea, abdominal pain, disturbances of vision)

Occasional: blood dyscrasia; photosensitivity

Rare: blindness

SODIUM SURAMIN USP (Germanin)

Frequent: vomiting; pruritus; urticaria; paresthesia; hyperesthesia of hands and feet; photophobia; peripheral neuropathy

Occasional: kidney damage; blood dyscrasia; shock

STIBOPHEN USP (Fuadin)

Adverse effects similar to effects of ANTIMONY POTASSIUM TARTRATE, but less frequent and usually less severe; additional effects are:

Occasional: heart damage with prolonged use

Rare: sulfhemoglobinuria; encephalopathy

SULFONAMIDES

Frequent: allergic reactions (rash, photosensitivity, drug fever); kernicterus in newborn

Occasional: renal damage; liver damage; Stevens-Johnson syndrome (more likely to occur with long-acting sulfonamides); blood dyscrasia; disseminated vasculitis

TETRACHLOROETHYLENE USP

Frequent: nausea; dizziness; headache

Occasional: liver damage; diarrhea

Rare: acute brain syndrome

TETRACYCLINES

Frequent: GI disturbance; bone lesions and staining and deformity of teeth in children up to 8 years old, and in the newborn when given to pregnant women after the fourth month

Occasional: malabsorption; enterocolitis; photosensitivity reactions (most frequent with demethylchlortetracycline); parenteral doses may cause serious liver damage; especially in pregnant women and patients with renal disease

Rare: allergic reactions; blood dyscrasia; interference with protein metabolism; increased intracranial pressure in infants; Fanconi-like syndrome from deteriorated tetracyclines

THIABENDAZOLE (Mintezol)

Frequent: nausea; vomiting; vertigo

Occasional: leukopenia; crystalluria; rash; disturbance of color vision

Rare: shock; tinnitus

TRYPARSAMIDE USP

Occasional: impaired vision; optic atrophy; fever; exfoliative dermatitis; allergic reactions; tinnitus; vomiting

Partial List of Brand Names

amodiaquine—Camoquin
amphotericin B—Fungizone
antimony sodium dimercaptosuccinate (stibocaptate)—Astiban (Hoffmann-La Roche, Switzerland)*
antimony sodium gluconate—Pentostam (Burroughs Wellcome, England)*
Bayer 2502 (a nitrofurfurylidene derivative)—(Bayer, Germany)*
bephenium—Alcopara
cycloguanil pamoate—Camolar (Parke, Davis)*
dapsone—Avlosulfon
dehydroemetine—Dehydroemetine (Hoffmann-La Roche, Switzerland)*
dichlorvos—Dichlorman (Shell)*
diethylcarbamazine—Hetrazan
diiodohydroxyquin—Diodoquin
lucanthone—Nilodin (Burroughs Wellcome, England)*
melarsoprol—Mel B (Rhone Poulenc, France)*
metronidazole—Flagyl
niclosamide—Yomesan (Bayer, Germany)*
niridazole—Ambilhar (Ciba, Switzerland)*
pentamidine—Lomidine (May & Baker, England)*
piperazine—Antepar
primaquine phosphate—Primaquine
pyrimethamine—Daraprim
pyrvinium pamoate—Povan
quinacrine—Atabrine
sodium suramin—Germanin (Bayer, Germany)*
stibophen—Fuadin
thiabendazole—Mintezol

*As of time of publication (March 1969), not available in the United States for general use. Information on availability may be obtainable from manufacturer or the Parasitic Disease Drug Service, Parasitic Diseases Branch, Epidemiology Program, National Communicable Disease Center, Atlanta, Georgia 30333; telephone 404 633-3311

HYPERACTIVE CAROTID SINUS REFLEX AND CAROTID SINUS SYNCOPE

Juergen E. Thomas, MD, Section of Neurology, Mayo Clin Proc 44(2):127–139, Feb 1969.

From the carotid sinus, a fusiform enlargement of the internal carotid artery at the level of the carotid bifurcation, arises an important physiologic mechanism that controls reflex regulation of cardiovascular function. Specialized nervous receptors embedded in the sinus wall constantly monitor systemic arterial blood pressure and act to maintain it within relatively narrow limits. These receptors constitute the sensing element in a reflex arc whose afferent limb is formed by the glossopharyngeal and probably vagus nerves and whose efferent limb is the cardiovascular autonomic outflow. The principal reflex mechanisms arising from the carotid sinus and other baroreceptor areas affect circulation, heart rate, and respiration. The undisputed changes elicited by an increase in intrasinusal tension are a fall in systemic arterial pressure, slowing of heart rate, and depression of respiration. Conversely, a decrease in tension within the sinus raises the blood pressure, accelerates the heart, and augments respiration.

The recognition that the carotid sinus reflex has not only physiologic but clinical implications as well is as old as the discovery of the reflex itself. Numerous accounts have appeared in the medical literature of instances of syncope and even convulsions in response to mechanical manipulation of the carotid sinus in man. Persons so afflicted were said to have a "hyperactive carotid sinus reflex." As a logical extension of this observation, the sinus reflex was studied as a possible cause for spontaneously occurring syncope and a relationship was shown to exist in some patients. Spontaneously occurring carotid sinus symptoms with or without syncope were referred to as the "carotid sinus syndrome."

The evaluation of patients with syncopal loss of consciousness is one of the commoner exercises in medical practice and an effort to determine whether the carotid sinus is causally responsible is often mandatory.

Since neither the mode of testing the carotid sinus reflex and assessing test results nor the clinical implications of the sinus reflex are clear to everyone, it seems appropriate to review both the hyperactive carotid sinus reflex and the carotid sinus syndrome

(carotid sinus syncope), placing emphasis on the sinus stimulation test, incidence of occurrence, clinical manifestations, precipitating factors, pathogenesis, and treatment.

Hyperactive Carotid Sinus Reflex

This term is applied when digital stimulation of the carotid sinus results in marked slowing of cardiac rate or in cardiac asystole accompanied by a fall in systemic blood pressure. In some cases only the blood pressure drops while cardiac rhythm remains unaffected. Opinions vary on how profound the cardiovascular changes must be to call the reflex pathologically hyperactive. Franke, who has studied the carotid sinus reflex extensively, regards cardiac asystole lasting 3 seconds or more and a decrease of systolic and diastolic blood pressure of 50 mm Hg or more as clearly abnormal, while a slowing of heart rate by 30 to 50 percent, cardiac asystole lasting 2 seconds, and decrease of 30 mm Hg in systolic blood pressure constitute a borderline response. These criteria represent a workable compromise and have been accepted widely.

The incidence of a hyperactive sinus reflex—as defined above—is extremely low in the first 3 decades of life but increases steadily thereafter. Males are affected twice as often as females. The reflex may be evoked easier from the right than the left carotid sinus; it may be present bilaterally.

Carotid Sinus Stimulation Test.—No agreement exists on the method to be employed for evoking the carotid sinus reflex. For years I have been using a slightly modified version of the technique described by Franke.

The test begins with a check for satisfactory pulsation of the carotid and temporal arteries and auscultation of the carotid vessels for bruits. If necessary, ophthalmic artery pressures are measured before the test. Patients with obvious cerebrovascular disease are tested under strict indications.

The patient is placed in the supine position, significant head rotation is avoided, and the head is kept in a neutral position between flexion and extension. The examiner's fingers feel gently for the fusiform carotid sinus or the area of greatest arterial pulsation lying in front of the sternomastoid muscle at the upper border of the thyroid cartilage. The carotid sinus is pressed lightly for 20 seconds while the examiner listens to the heart. An attending person palpates the temporal artery simultaneously and makes sure its pulsation is felt throughout the test. This maneuver is used as an indicator that distal carotid blood flow has probably not been compromised by the sinus manipulation. The right carotid sinus is stimulated first, followed by the left several minutes later. Never are both sinuses stimulated together.

If there is no unusual cardiac response, stimulus intensity is increased or the sinus is massaged gently in the longitudinal direction for 15 seconds. If again no response occurs, the procedure is repeated with the patient in the sitting position. While ideally the sinus stimulation test should be carried out under electrocardiographic control, this is impractical for routine bedside use.

If no cardiac response is elicited from either carotid sinus, the test is repeated and this time the blood pressure is recorded over the brachial artery at rapid intervals. This is again done with the patient supine to avoid complicating orthostatic factors but may have to be done in the sitting position as well.

If the carotid sinus is hypersensitive, heart or blood pressure response or both occur almost always in the first 15 to 20 seconds of the test. In my experience it is hardly ever necessary to stimulate for longer than 30 seconds. Fluctuations in sinus sensitivity do occur but more often than not successive tests in the same patient yield similar results.

The carotid sinus stimulation test is not without hazard. Complications have ranged from visual disturbance to hemiplegia to sudden death, the latter mostly in seriously ill patients or during anesthesia or operation. While some complications are unforeseeable, I believe with Franke that in many of the published cases of complications either the test was done carelessly or patients were subjected to it who should not have been. If the carotid stimulation test is performed in the most careful manner and is avoided in unsuitable patients, the likelihood of encountering serious complications is extremely small. This has been attested to in thousands of cases.

Clinical Manifestations.—A variety of symptoms and signs occur during the stimulation test in individuals with a hyperactive carotid sinus reflex. Their ease of induction, intensity, and duration are influenced by the position of the body, the age of the person, the status of the cerebral circulation, and the psychologic makeup of the individual.

Among the subjective manifestations, lightheadedness is the most common and often the initial symptom. Other frequent manifestations are faintness, blurring or darkness of vision, a feeling of general weakness, and pressure or fullness in the head. Also tinnitus, paresthesias in the hands, dyspnea, head-

ache, confusion, nausea, and sweating have been reported. These symptoms obviously are not specific and can be part of any other type of syncope as well.

In many cases of hyperactive carotid sinus reflex the above symptoms culminate in loss of consciousness, usually of sudden onset. This is immediately preceded or accompanied by pallor of the face, coldness of the skin, and deep respiration. During the faint the individual is usually motionless and limp, but occasionally when cardiac asystole is prolonged a few tonic or clonic jerks of limbs and face can be seen. These convulsions are neither clinically nor electrophysiologically related to grand mal seizures. A fleeting intense facial flush may accompany the convulsive movements. As in other types of syncope, if the bladder happens to be full and the patient falls abruptly, there may be urinary incontinence.

Unconsciousness is brief, lasting from seconds to a minute or two at the longest. Sensorium is quickly restored and the person feels well again. After a prolonged spell there may be short-lasting drowsiness or lassitude. To be productive of syncope, cardiac asystole must last anywhere from 5 to 15 seconds. The occurrence of syncope is an indication for immediate cessation of the stimulation test.

The cardiovascular response on digital stimulation of the carotid sinus was divided by Weiss and Baker into three types: (1) the cardioinhibitory type with bradycardia or asystole with or without systemic hypotension, (2) the vasodepressor type not associated with cardiac slowing, and (3) a primary cerebral type not accompanied by either systemic hypotension or bradycardia.

The cardioinhibitory type is by far the most common variety of the hyperactive carotid sinus reflex. Its incidence has been estimated to vary from 34 to 78 percent among persons with sinus hypersensitivity.

The pure vasodepressor type, on the other hand, is rare, making up only 5 to 10 percent of the cases of hyperactive sinus reflex.

Whether the so-called primary cerebral type exists is highly debatable. Doubtlessly, many of the cases which had previously been regarded as "cerebral" are in fact the result of mechanical interference of the carotid blood flow during the stimulation test when there is occlusive disease involving the opposite carotid, the anterior cerebral, or the vertebral-basilar arterial system. If the cerebral form of the carotid sinus hypersensitivity exists, it must be exceedingly rare and is clinically of little or no practical importance.

Division of symptoms according to the type of reflex hyperactivity serves no purpose. The symptom complex in the vasodepressor variety may follow a more protracted course than in the cardioinhibitory type but is otherwise the same. Moreover the various reflex types often occur in mixed form, though one usually predominates.

In the cardioinhibitory type, the cardiac manifestations depend largely on the reflex responsiveness of the heart at the time of the test. Cardiac asystole may last from 2 to 15 seconds. Electrocardiographic abnormalities include sinoatrial slowing, atrial conduction defects, prolongation of the P-R interval, atrioventricular block, sinoatrial arrest, nodal escape, complete asystole, and ventricular ectopic beats. Although Weiss and Baker state that cardiac asystole can occur without a fall in blood pressure, this seems highly unlikely. Certainly in longer-lasting asystole the systemic blood pressure does fall, but intra-arterial recordings may have to be used to demonstrate it.

Electroencephalographic studies have shown that during the initial 3 to 4 seconds of cardiac asystole the EEG is normal. After transient flattening of electric activity during which the patient complains of dizziness and looks pale, there may be increased beta activity. With prolonged asystole (7 to 15 seconds), associated clinically with unconsciousness, dilated pupils, and unobtainable blood pressure, EEG activity reveals diffuse slowing of activity in the theta and delta ranges followed by temporary flattening of the recording. With reawakening of cardiac activity, the physiologic EEG pattern returns in 10 to 15 seconds.

As noted above, the vasodepressor type of carotid sinus hypersensitivity is rare. The symptoms it causes are the same as in the cardioinhibitory type except that syncope probably never occurs in the recumbent position. The symptoms tend to follow a more protracted course because the blood pressure may not recover its normal level for minutes. However, symptoms and signs commence just as quickly as in the cardiac form, namely within the first 15 to 20 seconds of sinus stimulation.

Pharmacologic Identification of Type.—For further identification of the type of hyperactive carotid sinus reflex, pharmacologic substances have been used. If the stimulation test evokes cardiac manifestations and systemic hypotension, 1.0 mg of atropine may be given intravenously and the patient reexamined in 5 minutes. Disappearance of cardiac changes and clinical symptoms on renewed sinus

pressure presumably confirms that the previous test response was "cardioinhibitory or vagal" in type. Should the blood pressure still fall despite the use of atropine, epinephrine is given parenterally (0.5 ml of a 1:1,000 aqueous solution). If symptoms and hypotension are abolished, the patient is assumed to have the "vasodepressor" type of carotid sinus hypersensitivity.

Precipitating and Predisposing Factors.—The carotid sinus reflex is especially prone to be abnormally active in the presence of cardiovascular disease. Arteriosclerosis, hypertension, and coronary artery disease all predilect to sinus hypersensitivity. The same occurs in digitalized patients. In fact, digitalis intoxication may be considered in a digitalized patient who develops advanced degrees of heart block during carotid sinus stimulation, especially when this response was absent previously. Also diseases involving the afferents to the vagus center may lower the reflex threshold of the carotid sinus as has been demonstrated in patients with acute disease of the biliary tract. Another condition said to be associated at times with sinus hyperactivity is diabetes mellitus. Treatment of diabetics with insulin may increase the sensitivity even further.

Local pathologic changes adjacent to the carotid sinus in the neck, such as enlarged lymph nodes, tissue scars, and rarely carotid body tumors, are known to cause a hyperactive reflex. Also patients afflicted with Takayashu's disease (pulseless disease), a specific form of progressive obliterative brachiocephalic arteritis, may suffer from sinus hypersensitivity.

Carotid Sinus Syndrome (Carotid Sinus Syncope)

While the hyperactive carotid sinus reflex has limited clinical significance and does not give rise to spontaneous symptoms, the carotid sinus syndrome (also called spontaneous carotid sinus syncope) is clinically significant and should be considered separately, for symptoms are now occurring spontaneously. Most individuals exhibiting abnormal sinus sensitivity on manual testing do not suffer spontaneous sinus syncope. Conversely, patients with spontaneous syncope usually do have a hyperactive reflex. It is not easy to reject the claim that the two are closely related, differing perhaps only in matters of severity. By definition, the diagnosis of spontaneous carotid sinus syndrome is said to be established if spontaneously occurring symptoms of dizziness, blurring of vision, faintness, or fainting can be reproduced by stimulation of one or both carotid sinuses, provided carotid blood flow is preserved during the test.

Carotid sinus syncope is a rare condition. Some 5 to 20 percent of persons with a hyperactive reflex are said to have it. As in the hyperactive reflex, carotid sinus syncope occurs more often in men than in women and is commoner in the older age groups, especially the sixth and seventh decades.

Clinical Manifestations.—The symptoms occurring in the spontaneous syndrome are the same as those that occur as a result of the hyperactive reflex. They have been described above and need not be recounted. The attacks happen for the most part in the sitting or standing position and tend to follow a similar course in the individual patient, but changes in symptom content and in severity from attack to attack are known to occur. Frequencies of several episodes a day to one every few months have been reported.

The spontaneous syndrome—like the hyperactive reflex—has been divided into the cardioinhibitory, the vasodepressor, and the cerebral type. This division is nearly always made on the basis of the patient's response to the sinus stimulation test, since one rarely has the good fortune of being able to study the patient during one of his spontaneous attacks.

Precipitating and Predisposing Factors.—In most patients suffering from spontaneous carotid sinus syncope, no obvious cause for the spells can be identified. However, when there are trigger mechanisms, these may be of extravascular or intravascular origin.

Among the extravascular factors, forced head-turning when backing up a car, hyperextension of the neck when looking at the sky, viewing a movie from the front row, working overhead, and the like are said to have been responsible for the syncope. Other local trigger factors have included pressure of constricting neckwear, stretching of the skin over the neck when shaving, and carrying heavy shoulder loads. It should be remembered, however, that forced positional changes of the head are known to also cause syncope or clouding of consciousness by strangulation of blood flow through the cervical vertebral arteries.

Pathologic changes adjacent to the carotid sinus such as thyroid tumors, carotid body tumor, and inflammatory and malignant lymph nodes are other extravascular conditions believed to have precipitated the spontaneous syndrome just as they are blamed for some cases of hyperactive carotid sinus reflex.

In certain predisposed individuals, intravascular excitation of the carotid baroreceptors can appar-

ently also result in spontaneous symptoms. Straining at stool, heavy lifting, and strong coughing have reportedly produced sinus syncope. Whether these cases could withstand critical appraisal is another question.

Diagnosis of the Carotid Sinus Syndrome.—The carotid sinus syndrome is diagnosed on the basis of (1) spontaneously occurring episodes of syncope, often preceded by dizziness, weakness, and faintness, and (2) the reproduction of the same symptom complex on digital stimulation of the carotid sinus but not on pressure on the carotid artery elsewhere.

Arriving at a diagnosis in this fashion is unsatisfactory. First, there is nothing unique about the symptom quality of the carotid sinus syndrome. The presyncopal discomfort is as nonspecific as the syncope itself. Although the abrupt onset of symptoms will alert the physician to a possible reflex nature of the syncope, the carotid sinus is just one of several sites from which reflex syncope may arise.

Second, to regard the reproduction of symptoms on digital sinus stimulation as diagnostic confirmation of the carotid sinus etiology of the spontaneous syndrome is highly speculative. The hyperactive carotid sinus reflex occurs much more often than the spontaneous syndrome and the presence of an overactive reflex in a patient with spontaneous faints may therefore be merely coincidental. A history of repeated syncope clearly related to activities that press or stretch the sinus in a patient who also has a positive reaction on carotid stimulation is diagnostically helpful. However, most patients do not furnish such a history. If spontaneous syncope and hyperactive sinus reflex coexist in a patient and therapeutic measures directed at the carotid sinus abolish both, a common etiologic bond may be assumed to have been present.

In contrast to the claim that the diagnosis of carotid sinus syncope can be established with ease, it is my experience that an unequivocal diagnosis is made with difficulty. Usually the diagnostic evidence is presumptive.

Pathogenesis of Carotid Sinus Syncope.—The lesion causing hyperactivity of the carotid sinus reflex can be situated anywhere along the course of the reflex arc. Preferential sites are believed to be the receptor region at the carotid sinus, the reflex centers in the brain stem and hypothalamus, and the target organs themselves. There is no completely satisfactory explanation for the presence of spontaneous symptoms in some persons and their absence in others.

A detailed discussion of the pathogenesis will be omitted. It can be found elsewhere. One of the most attractive theories is that abnormalities at multiple sites in the carotid reflex arc join in the production of the hyperirritability. Franke views a combination of sclerotic changes in the carotid sinus and coronary vessels as the leading cause for the cardioinhibitory type of sinus sensitivity; changes at only one of these two sites will not produce reflex abnormality. A greatly overactive reflex in persons older than 40 years suggests to Franke the presence of coronary sclerosis even when other clinical evidence is lacking.

Treatment of Carotid Sinus Syncope.—Since there are no spontaneous symptoms, a hyperactive carotid sinus reflex requires no treatment.

In patients with spontaneous but infrequent carotid sinus syncope an explanation of the problem and reassurance are sufficient. When frequency of occurrence becomes significant, a careful search for remediable precipitating factors should be undertaken. Focal lesions about the carotid sinus should be removed and trigger mechanisms (tight neckwear and the like) avoided. Drugs known to sensitize the vagus are best used only under strict indications.

In cardioinhibitory sinus syncope, parasympathicolytic drugs such as atropine sulfate given orally in doses of 0.5 mg (1/120th grain) three or four times a day may be tried. These agents should be avoided in patients with latent or manifest glaucoma. The rare vasodepressor sinus syncope may respond to oral administration of ephedrine in doses of 15 to 30 mg (¼ to ½ grain) or amphetamines.

Severe carotid sinus syncope requires energetic treatment measures. Conventional drug therapy is usually fruitless in these cases. No agreement exists whether radiation therapy to or surgical denervation of the carotid sinus should be given preference. Since there is little difference between the two in incidence of improvement or cure and since surgical treatment is not without risk, it seems advisable to try irradiation before operation.

Opinions vary on the amount of radiation therapy to be given. A single dose of 400 r, measured in air, to one side of the neck, a total dose of 500 r if unilateral and 400 r if bilateral, or a total dose of 600 r (200 r every other day) to each carotid sinus has been mentioned. It seems that radiation therapy provides good to excellent results in about two thirds of patients with spontaneous sinus syncope of either the cardiac or the vasodepressor type and does so quickly and apparently without side-effects.

Surgical denervation of the carotid sinus has been used in selected patients. Because of the rise in systemic blood pressure which commonly occurs postoperatively and may last for hours, days, or weeks, the procedure is hazardous to hypertensive patients, especially those known to have cerebrovascular disease. A preoperative block of the carotid sinus nerve with a local anesthetic agent is advisable. Although this will not allow prediction regarding the effect of surgical treatment on the carotid sinus syncope, it may provide information concerning the surgical effect on the hyperactive sinus reflex. In patients with bilateral sinus hypersensitivity and spontaneous syncope, operation may be restricted to the side of greater sensitivity. This may reduce sinus irritability to a level where drug therapy may be effectively instituted.

In place of surgical denervation of the carotid sinus, intracranial sectioning of the glossopharyngeal nerve has been carried out. This interrupts the afferent limb of the sinus reflex just as effectively and does not usually leave a demonstrable neurologic deficit.

A Note on Differential Diagnosis.—For details concerning the differential features of fainting, the reader may wish to consult other publications. Only a few remarks are in order here.

None of the clinical manifestations comprising the syncopal syndrome is sufficiently distinctive to provide conclusive evidence of the underlying cause. However, in many cases valuable information can be gleaned from a careful analysis of such factors as the degree of fatigue and emotional tension of the patient, recent illness, the environmental temperature, the age of the patient, the position of the body, and the activity engaged in at the time of the faint, the duration as well as the manner of onset of and recovery from the spell, and associated symptoms and signs.

At a young age simple vasodepressor syncope—the classic benign faint—outranks all others in frequency of occurrence. Rare other causes at this age are congenital cardiac anomalies, valvular heart disease, and paroxysmal tachycardia. In later adult life vasodepressor syncope occurs less frequently and organic conditions such as intrinsic heart disease, orthostatic hypotension, carotid sinus hypersensitivity, and cerebrovascular disorders are more common.

In the simple vasodepressor faint, presyncopal symptoms are often pronounced and prolonged, while sudden loss of consciousness with little or no warning may occur in primary or reflex cardiac asystole or severe orthostatic hypotension. In the latter, syncope is precipitated by postural changes of the body to the upright position but also at times by exercise. A faint occurring in recumbency is nearly always the result of prolonged cardiac asystole. Loss of consciousness following physical exertion has been observed in coronary heart disease, aortic stenosis, congenital cardiac anomalies, primary pulmonary hypertension, orthostatic hypotension, and Takayashu's disease (or less specifically, the aortic arch syndrome).

In final analysis it appears that, in the interpretation of brief episodes of unconsciousness, the total picture of the attack is often more important than the presence or absence of any specific symptom. It should be remembered that syncope, although for the most part a benign and transitory condition, occasionally is the manifestation of a serious organic disease. A careful and systematic search for such underlying disease is therefore mandatory when syncope grows into a repetitive and disturbing event. While carotid sinus syncope is an infrequent disorder, it is treatable and should not go undetected.

(The references may be seen in the original article.)

TINEA INCOGNITO

F. Adrian Ive, MB MRCP; Ronald Marks,* MB BSc MRCP DTM&H, Reprinted from the British Medical Journal, 1968, 3, 149–152, by permission of the Authors and Editor.*

Summary: Fourteen cases are described in which the local application of corticosteroid preparations to ringworm infections of the skin have resulted in unusual clinical pictures. A kerion-like lesion due to Trichophyton rubrum, intertriginous infections simulating candidiasis and due to Epidermophyton floccosum, and pictures resembling poikiloderma, papular rosacea, and indeterminate leprosy are among the changes that were seen in these patients.

Introduction

Dermatology has been greatly helped in recent years by the introduction of a variety of potent therapeutic agents. While this has often meant that diseases are easier to control it has also resulted in an increase of iatrogenic disease.

Corticosteroid ointments tend to be used as a dermatological panacea and the misuse of these powerful agents is often the cause of commonly observed iatrogenic skin disease. While the frequently disastrous results of the systemic abuse of corticosteroids have been well documented there are few references to the problems that result from their inappropriate topical application (Grice, 1966).

Dermatologists have become increasingly aware that the clinical appearance of some rather common skin diseases may be rendered almost unrecognizable by topical steroids and particularly by the use of their fluorinated derivatives. This applies especially to those dermatoses in which the use of these compounds is normally contraindicated.

A principal action of corticosteroids is to suppress inflammation, and when administered systemically they can hinder immune responses. In this way they may contribute significantly to the morbidity of infective disease of all types, specific examples being bacterial infections such as tuberculosis, viral infections such as chicken-pox, and fungal diseases such as ringworm (Kligman, Baldridge, Rebell, and Pillsbury, 1951). Extensive tinea corporis is a well-documented complication of Cushing's disease (Canizares, Shatin, and Kellert, 1959). Thus it is surprising to find that corticosteroids are not infrequently used in the management of infective skin disease. This situation probably derives in part from the tendency of patients to indulge in self-medication with hoarded ointments. In most instances, however, it would appear to stem from the recognition, by doctors, of the undoubted ability of steroids to provide prompt relief of symptoms by the suppression of inflammation. Systemic immune responses can usually contain the infection, and patient satisfaction is assured. This appertains especially in herpes simplex, when the risk of promoting keratitis is often disregarded.

Impetigo, scabies, and specifically ringworm infections are also often mistakenly treated by local corticosteroid preparations and sometimes even by systemic administration. The bizarre clinical pictures which ensue can tax the most expert diagnostician. The following cases illustrate the difficulties resulting from the use of local corticosteroids in the treatment of superficial fungus infections.

Case Reports

Case 1.—A West Indian woman aged 31 complained of an irritating rash that started in the groins and spread to the adjoining thighs. She had been given Synalar (fluocinolone acetonide) and Betnovate (betamethasone-17-valerate), which brought transient symptomatic relief only. On examination there were well-defined patches which were non-scaly, depigmented, purplish, and telangiectatic. Fungal mycelium was seen in a scraping of the involved skin and *Epidermophyton floccosum* was grown from skin scales. She was treated with half-strength Whitfield's ointment and was clear after three weeks.

Case 2.—A woman aged 71 had a rash on the feet and ankles for the previous year. She had used betamethasone under polyethylene occlusion. The rash was red and scaly and had a well-defined edge. It extended over the occluded area like a pair of socks. *Trichophyton rubrum* was grown and she was much improved after four weeks' treatment with griseofulvin and Whitfield's ointment.

Case 3.—A 23-year-old man had since the age of 17 had various itchy rashes diagnosed as seborrhoeic dermatitis. On examination there was a macular plum-red non-scaly rash around the right eye, in the

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groins, and on the sides of the neck, with poorly defined margins. He had used fluocinolone ointment and other local steroid applications. An examination for fungus showed the presence of ringworm type mycelium in the skin around the eye and on the groins. *E. floccosum* was grown from all areas. His rash cleared after one month on griseofulvin and Tinaderm (tolnaftate) cream.

Case 4.—A 53-year-old woman had had lichen simplex on the right forearm and elbow since 1955, which was controlled by the use of fluocinolone ointment. In January 1966 she developed a rash in the groins, under the breasts, and in both axillae which was bright red, scaly, and cracked in places, with tiny outlying satellite pustules which suggested candidiasis. There was also scaling between the toes. She used betamethasone to each patch as it developed. Microscopical examination of scales showed a ringworm fungal mycelium, and E. floccosum was grown from all sites. She was treated with griseofulvin and undecenoate ointment, and had much improved after six weeks.

Case 5.—A woman aged 45 complained of a rash under the breasts and in the axillae for the previous six months. The clinical diagnosis was candidal intertrigo, but scaling was noted in the toe webs. She had used various preparations, including Synalar-N. Microscopical examination showed a ringworm fungal mycelium in all sites and *E. floccosum* was cultured. She was treated with half-strength Whitfield's ointment topically and griseofulvin and was clinically clear in less than three months.

Case 6.—A man aged 25 had had a rash in the groins for the previous three months and multiple boils for the previous five or six weeks. He had used many proprietary steroid preparations to the area in the groins. On examination there was a well-defined mauvish rash in the groins. Its surface was slightly scaly and telangiectatic. Ringworm fungus mycelial filaments were seen in scrapings from both groins and toe webs. Complete clearing occurred after six weeks on oral griseofulvin and local half-strength Whitfield's ointment.

Case 7.—A 19-year-old female laboratory technician was given betamethasone cream by her practitioner for spots on the legs diagnosed as insect bites. No improvement resulted after four months of this treatment. When seen in the dermatology clinic there was an indurated area below the left knee in which prominent erythematous plugged and excoriated hair follicles were clearly visible. The clinical diagnosis was "folliculitis," and only on mycological examination could the correct diagnosis of tinea corporis be made. *T. rubrum* was grown from this area and the lesion cleared after five weeks' treatment with griseofulvin 250 mg twice daily and Castellani's paint locally.

Case 8.—A 67-year-old man had a two-year history of intense irritation over the upper chest wall and buttocks. His doctor had treated him with Remiderm (triamcinolone acetonide with halquinol), betamethasone, fluocinolone, and Ultralanum (fluocortolone 21-hexanoate). On examination there was an extensive rash over the upper part of the front of the chest and root of the neck. It was light tan in colour, flat, non-indurated, and could be seen only with difficulty. The lesion on the buttocks was also smooth and non-scaly, but the light red colour and its serpiginous border suggested ringworm. *T. rubrum* was grown from both areas and from the soles of the feet. He was cleared after one month's treatment with griseofulvin 250 mg twice daily and local Whitfield's ointment.

Case 9.—A youth aged 18 was diagnosed as having "dhobie itch" by his doctor but was given Propaderm (beclomethasone dipropionate) and fluocortolone to apply to the affected area. The rash altered in appearance during the next three months but the itching persisted. On examination he had broad mauve striae distensae on the inner aspect of the upper thighs. No scaling was evident and the surrounding skin looked clinically normal. Microscopical examination of skin scales showed a ringworm fungal mycelium. His symptoms subsided on griseofulvin and tolnaftate cream but the striae persisted.

Case 10.—A youth aged 19 gave a six-month history of rash in the groins which had been treated with fluocinolone and betamethasone. On examination a confluent and vivid erythematous rash was seen. On closer inspection there was an appearance of atrophy, scaling was minimal, and many telangiectases were seen within the area. Ringworm fungal mycelium was found in scrapings from the site. The lesion responded to treatment with Whitfield's ointment.

Case 11.—A 50-year-old man developed red rings on his face three months previous to being seen in the clinic. A dermatologist had diagnosed erythema annulare centrifugum. This seemed to respond to treatment with oral antihistamines and local betamethasone cream. After six weeks of this treatment he developed an inflammatory lesion on the left side of the upper lip. When seen two weeks later he had

developed an indurated area studded with pustules and partially covered with a yellow crust on the upper lip. There was also faint erythema and scaliness on the soles suggestive of tinea pedis. The lesion on the lip was diagnosed as kerion clinically and a large spored ectothrix type of fungus was seen microscopically. *T. rubrum* was grown both from the kerion and the soles. He was treated with griseofulvin 125 mg four times a day and with Whitfield's ointment locally. He was completely cleared after three months.

Case 12.—A 66-year-old woman with a 40-year history of psoriasis attended with a four-month history of a reddened eruption on the antecubital fossae, to which she had been applying betamethasone without effect. The areas were found to be red and atrophic with multiple fine telangiectases. Clinical diagnosis of atrophic morphoea and poikiloderma were entertained. The diagnosis of ringworm was made only on biopsy examination after finding mycelia in the stratum corneum. *E. floccosum* was grown from skin scrapings from the area and she responded completely to a month's course of griseofulvin and Whitfield's ointment locally.

Case 13.—A woman aged 39 presented with a nine-month history of a spreading rash on the face. She had had a rash on the left palm for the previous two years which was irritant, red, and slightly scaly and which cracked in the cold weather. The rash on the face started at the corner of the mouth and spread outwards; it was red, non-scaly, and there were many minute flat-topped papules. Though the eruption was diffusely distributed over the face a clinical diagnosis of rosacea had been suggested. She had been treated with betamethasone and many other local steroid preparations. A mycological examination confirmed the presence of abundant fungal mycelium in scrapings from the face and the hand.

Case 14.—A 28-year-old Indian man had had a rash on his hand for many years. For two months he had noted a rash on his face and applied fluocinolone and fluocortolone regularly. On examination there was a marginated hypopigmentation of the whole front of his face with minimal scaling and no erythema. Sensation was normal and a biopsy specimen taken to exclude leprosy showed eczematous changes only. At this stage the fairly obvious fungal lesion on the hand was noted and ringworm mycelia were seen microscopically in scrapings from both areas. He subsequently cleared on griseofulvin and Tinaderm.

Discussion

From the clinical material presented above some points of interest arise. In general the clinical pictures that resulted from the application of topical corticosteroids to ringworm infections were bizarre and difficult to recognize, but in one patient (Case 2) it was typical in appearance though of unusual extent.

Ringworm fungi metabolize dead keratin, and their presence in the horny layer evokes an eczematous response in the viable epidermis beneath. Eczematous skin is a poor producer of keratin and so, deprived of foodstuff, fungous infections tend to resolve (Pillsbury, Shelley, and Kligman, 1956). It can be seen that topical steroids, by suppressing the eczema, may encourage fungal growth. In addition apparent enhancement of the virulence of ringworm organisms by suppression of local immune responses may occur. A similar situation has recently been noted in which local overgrowth of candida due to steroid applications has complicated oral lichen planus (Cawson, 1968) and otitis externa (R. A. Williams, personal communication, 1968), where eradication can prove extremely difficult. The appearance of tinea after topical application of such potent steroids as fluocinolone acetonide or betamethasone-17-valerate is probably determined, in varying proportions, by the above two considerations.

Apparent increased virulence resulting in eruptions in unusual sites and of unusual extent and appearance was seen in Cases 4 and 5 where involvement of intertriginous areas simulated candidiasis, and also in Cases 3, 13, and 14 where non-flexural skin became involved. An extreme degree of this artificially boosted virulence is exemplified by Cases 7 and 11, where *T. rubrum* invaded hair follicles to cause kerion and folliculitis. This fungus has very rarely been implicated in kerion formation. The local action of corticosteroid preparations in producing striae distensae in sites of application is well recognized (Epstein, Epstein, and Epstein, 1963; Meara, 1964), and Case 9 is an example of this reaction.

Case 10 illustrates a feature that was seen in several of our patients which does not appear to have been described before and will be the subject of a fuller communication (Munro, 1968). The affected area in these patients showed atrophy and telangiectasia without scaling, giving the skin a translucent and poikilodermatous appearance. In Case 12 this

occurred on the arm and gave rise to extreme diagnostic difficulty, but in Cases 1, 3, 6, and 10 groin involvement suggested the correct diagnosis.

Hypopigmentation was seen in both coloured patients (Cases 1 and 14). This phenomenon does not usually follow resolution of ringworm after orthodox treatment and was confusing in Case 14, where a diagnosis of leprosy was initially entertained.

Case 13 was seen by several dermatologists before the correct diagnosis was made. The micropapules on her face were more suggestive of a type of rosacea than ringworm, and only the recognition of modified tinea elsewhere on her body suggested the correct diagnosis.

In the present context the old adage of diagnosis before treatment is probably impracticable in most general practices. It should be more widely known that skin scrapings may be taken for mycological examination as easily as bacteriological swabs. None the less it is likely that cases of masked ringworm will continue to occur. In our opinion the best form of prophylaxis is an increased awareness of tinea, especially in cases of groin and unilateral hand eruptions.

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(The figures and references may be seen in the original article.)

ALCOHOL, ASPIRIN, AND GASTROINTESTINAL BLEEDING

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Summary: In 20 healthy male subjects faecal blood loss was measured by means of a chromium-51-labelled red blood cell technique. Mean daily faecal blood loss associated with unbuffered aspirin ingestion was significantly increased by alcohol in the 13 subjects studied. In seven others alcohol alone did not cause gastrointestinal bleeding. These findings suggest that alcohol may accentuate gastrointestinal blood loss associated with unbuffered aspirin ingestion.

Introduction

It is a general clinical impression that aspirin and alcohol may be associated with erosive gastritis. Whereas there have been many investigations of the effect of aspirin on gastrointestinal blood loss and it is well established that unbuffered aspirin increases such loss (Smith and Smith, 1966), there have been no published studies of the effect of alcohol (ethanol) on gastrointestinal loss of blood. Furthermore, there have been no studies of gastrointestinal blood loss when alcohol and aspirin were taken together.

In the present investigation gastrointestinal blood loss was measured after the ingestion of aspirin alone, aspirin plus alcohol, and alcohol alone. It was found that alcohol did not itself cause gastrointestinal blood loss, but it did augment aspirin-induced gastrointestinal blood loss.

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Methods

Studies of faecal blood loss from the ingestion of aspirin alone and aspirin plus alcohol were made in 13 healthy male volunteers. In seven other subjects alcohol alone was ingested. The ages of the 20 subjects ranged from 20 to 25 years and none had a history of gastrointestinal disease. Gastrointestinal blood loss was estimated by the well-established technique of labelling the red blood cells of each subject with sodium chromate-51 and counting radioactivity in the faeces (Goulston and Skyring, 1964). Collection of faeces was started 48 hours after labelling, a disposable paint tin being used for each 24-hour faecal specimen. Faeces were collected for 20 consecutive days. On the 6th, 7th, and 8th days, after a six-hour fast, seven tablets of soluble unbuffered aspirin (Disprin) were taken by each subject as follows: one tablet (300 mg of aspirin in each tablet) at 6 p.m., one at 6:30 p.m., one at 7 p.m., two at 10 p.m., and two at 7 a.m. the following day. On the 14th, 15th, and 16th days seven tablets of aspirin were taken in the same fashion and alcohol was also

administered. Alcohol, in the form of undiluted Australian whisky (31.8% w/v ethanol), was taken as follows: 60 ml at 6 p.m., 60 ml at 6:30 p.m., and 60 ml at 7 p.m.—a total of 180 ml on each of the three days. The order of administration was reversed in 6 of the 13 subjects; thus aspirin plus alcohol was taken on the 6th, 7th, and 8th days, and aspirin alone on the 14th, 15th, and 16th days.

The effect of alcohol ingestion alone was studied in seven other male subjects. Faeces were collected for 12 consecutive days and alcohol was taken as described above on the 6th, 7th, and 8th days.

Specimens were counted in a high geometry counter having a liquid phosphor (ARMAC., Packard Instrument Co., Illinois). For the purpose of statistical comparison the first part of the study was divided into three six-day periods—days 1 to 6, days 7 to 12, and days 15 to 20. In the experiments using alcohol alone days 1 to 6 served as control and days 7 to 12 as the experimental period. These divisions were made to allow for the usual delayed appearance of blood in the faeces from upper gastrointestinal bleeding. The mean daily faecal blood loss in each of these periods was calculated by dividing the total blood loss in each period by six. The difference in faecal blood loss during control and experimental periods was evaluated by the *t* test (Snedecor and Cochran, 1967).

Results

In each of the 13 subjects the mean daily faecal blood loss was increased after aspirin ingestion (Table I). Mean daily faecal blood loss for all 13 subjects during the control period was 0.4±0.04 ml, during the aspirin period it was 3.2±0.6 ml. In response to alcohol plus aspirin the faecal blood loss in 11 of the 13 subjects was greater than that during the aspirin period. Mean daily faecal blood loss in response to aspirin plus alcohol (5.3±0.5 ml) was significantly greater than that in response to aspirin alone ($P<0.01$).

TABLE I.—*Effect of Aspirin and Aspirin Plus Alcohol on Mean Daily Faecal Blood Loss (ml./Day)*

Subject	Control Period	Aspirin Period	Alcohol + Aspirin
1	0.4	1.4	4.5
2	0.2	0.7	2.7
3	0.6	4.9	5.0
4	0.4	3.4	7.8
5	0.3	2.3	3.7
6	0.3	5.7	4.2
7	0.4	1.3	7.5
8	0.2	2.7	7.5
9	0.2	2.4	2.9
10	0.5	2.8	6.8
11	0.5	3.8	6.2
12	0.2	8.5	7.0
13	0.4	1.3	3.3
Mean	0.4	3.2	5.3
S.E.M.	0.04	0.6	0.5

In the seven other subjects to whom alcohol alone was given there was no significant increase in the mean daily faecal blood loss with alcohol ingestion (Table II).

TABLE II.—*Effect of Alcohol on Mean Daily Faecal Blood Loss (ml./Day)*

Subject	Control Period	Alcohol Period
14	0.2	0.3
15	0.2	0.3
16	1.8	0.6
17	0.2	0.2
18	0.4	0.6
19	0.2	1.9
20	0.2	0.8
Mean	0.5	0.7

Discussion

Although there has been extensive investigation of the various effects of alcohol in man, no studies of the effect of alcohol on gastrointestinal blood loss have been published.

In the present investigation gastrointestinal blood loss after ingestion of alcohol did not differ from that during the control period in the seven subjects studied (Table II). Leonards (personal communication, 1968) has also been unable to produce gastrointestinal bleeding in normal subjects after ingestion of alcohol. It is possible, however, that the concentration of alcohol in contact with gastric mucosa may be a critical factor.

In the present study alcohol when given alone did not cause gastrointestinal bleeding, but in the same dosage it increased aspirin-induced gastrointestinal blood loss (Table I). Gastrointestinal blood loss induced by unbuffered aspirin is variable from individual to individual, but Croft and Wood (1967) found that the bleeding response of each individual subject was reproducible. They studied gastrointestinal bleeding due to aspirin ingestion in 21 subjects on two occasions and found the mean daily faecal blood loss to be 6.3 and 6.5 ml. Thus in the present study the statistically significant increase in gastrointestinal blood loss associated with ingestion of alcohol

plus aspirin was almost certainly due to the addition of alcohol and not to variation in bleeding in response to aspirin alone.

The mechanism of action of alcohol on gastrointestinal blood loss induced by aspirin was not studied in the present experiments. Davenport (1965) found that in dogs with a Heidenhain pouch aspirin caused bleeding from the pouch only if the *p*H of the contents was acid. As alcohol is a known stimulant of gastric acid secretion it is possible that the increased bleeding with alcohol plus aspirin was a result of lowered *p*H of the gastric contents during the period aspirin was in the stomach.

We are grateful to Miss Angela Birchall and Miss Irena Lopata for technical assistance, to the subjects for their co-operation, and to Mr. David Paix, physicist, Prince Henry Hospital, for advice and for the use of the ARMAC counter. One of us (K. G.) was supported by an Abbott Fellowship of the Royal Australasian College of Physicians, and another (A. R. C.) by the National Health and Medical Research Council, Australia. This work was supported in part by the Bushell Trust.

(The references may be seen in the original article.)

RESERVE SECTION

NAVAL RESERVE HOSPITAL CORPSMEN COMPLETE REFRESHER TRAINING

A group of twenty-six Naval Reserve Hospital Corpsmen and three Coast Guard Corpsmen completed a two-week intensive course of instruction during the period 17 August to 30 August 1969 at the Naval Hospital, St. Albans, N.Y. This was the fifth such course at St. Albans under the direction of Master Chief Hospital Corpsman Emanuel S. Ratner, USNR, NR Surface Division 3-63(L), NRTC, Freeport, N.Y., who originated the class in 1966. For this session Reservists came from all over the FIRST, THIRD, FOURTH, and FIFTH naval districts with the greatest number being from THIRD naval district training centers. Among the trainees many vocations were represented ranging from school teachers, medical technologists, barber, research scientists, salesmen, hospital unit manager, contact lens consultant, letter carrier, doughnut shop proprietor, student nurse, clerks, super-market manager, telephone company switchman, and one corpsman doing recruiting duty on temporary active duty.

CAPT G. H. Tarr, Jr., MC USN, Commanding Officer of the Naval Hospital, welcomed the reservists personally and conducted a personnel inspection at which he found the men "very sharp" and "outstanding." (See photo on page 37.)

The course stressed independent duty techniques and emphasized subjects that cannot be taught at the training centers. Much of the time was devoted to problems and diseases of Southeast Asia. Corpsmen, Medical, Medical Service Corps and Nurse officers, most of whom were Vietnam war veterans, contributed first hand information to the program of instruction.

Supplementing the many lectures, tours, and demonstrations, practical instruction was afforded through evening watches in the laboratory, pharmacy, admission room, security patrols, and wards. The collective interest, enthusiasm, and eagerness of the hospital staff in providing instruction and information demonstrated the value of organizing a group to report on ACDUTRA at one time. Instructor training sessions were also conducted and the corpsmen left St. Albans well prepared to acquaint fellow corpsmen at their local training centers with the knowledge acquired.

HMCM Ratner, was given able assistance by the Hospital In-Service Training Dept., headed by LCDR Joan MacEnery, NC USN, who assigned instructors for the sessions and arranged all ward tours of the hospital.

CAPT G. H. Tarr, Jr. inspects the Naval Reserve Hospital Corpsmen.—(Official U.S. Navy Photograph)

HOSPITAL ADMINISTRATION SECTION

CAUTION SUGGESTED WITH SPRAY OVEN CLEANERS

Underwriter's Laboratories warns volume feeders that spray-type oven cleaners can damage an oven as well as cause fire or eye damage, report the co-operative extension specialists at South Dakota State University. Arcing is the jumping of electricity from one point to another. It occurs when spray cleaners are directed at electrical contacts. This may cause short circuits and grounds.

Knob and push electrical controls or switches are especially susceptible to arcing, the specialists report. The cleaners can cause inaccurate oven temperatures if allowed to build up on the oven's thermostat. Ovens should be protected by keeping the spray of cleaners away from all electrical contacts, the South Dakota State Specialists advise.

RATION DATA

Ration statistics for total hospital food service program in Fourth Quarter and yearly averages for FY 1969 are as follows: (Source—Food Service Performance Analysis, NAVMED 1412).

	4th Qtr FY 1969	*Yearly total FY 1969*
Total rations served.....	1,465,860	5,833,978
Total cost of provisions.....	$1,965,693.00	$7,681,113.70
Average cost of ration (raw food or net cost)....	$1.340	$1.317
Average cost of whole, fresh milk/gallon.....	$0.82	$0.81
Average ounces served whole, fresh milk/ration..	26	26
Percentage of total expenditures for: Meat, fish and poultry....	37%	37%

	4th Qtr FY 1969	Yearly total FY 1969
Whole, fresh milk.....	11%	12%
All other categories.....	52%	51%
Average ration cost for hospitals by group:		
Group A (CONUS) 329,348 to 862,520 rations/yr.....	$1.326	$1.300
Group B (CONUS) 125,543 to 227,757 rations/yr.....	$1.314	$1.299
Group C (CONUS) 37,357 to 135,109 rations/yr.....	$1.397	$1.360
Group D (OCONUS) 34,798 to 293,875 rations/yr.....	$1.377	$1.373
Average % of attached inpatients served.....	77%	75%
Average % of attached staff/support personnel served.....	59%	58%
Average % of modified diets to total inpatients served.....	14%	16%
Average % of total expenditures for supplemental nourishments.....	2.5%	2.2%

RESEARCH SECTION

RESEARCH DIVISION CODE 71—BUMED

The Research Division is tasked with the promotion, administration, and coordination of the medical research and development program of the Navy. It maintains continuing liaison with other Bureau divisions on matters of mutual concern in order to keep advised of operational problems, to stimulate interest in and awareness of research and development capabilities, and to cooperate in the development of research and development proposals. Both basic and applied research and development are involved and include the biological, medical, and psychological sciences.

Research and development proposals, whether suggested by other divisions, received from field activities, or submitted by prospective civilian contractors, are evaluated for approval, modification, or disapproval by the Research Division. In accordance with operational requirements promulgated by the Chief of Naval Operations and by program guidance and planning material issued by the Chief of Naval Research, the Division also suggests to field activities potentially profitable avenues of research and development in terms of present or anticipated naval needs. An added responsibility of the Research Division is coordination between the various naval medical research activities and the medical research and development programs of the Navy Department, Department of Defense, other Federal agencies, and with certain agencies of approved foreign governments.

The productivity and accomplishments of the Navy Medical Department's research and development program has gained increasing recognition, an indication of which can be seen in the tremendous rise in authorized funds and assigned research and development projects. From 1965 to 1969 funding increased almost threefold, while research and development projects were more than doubled.

LIST OF RECENT PUBLICATIONS FROM RESEARCH LABORATORIES

The following papers have been completed by research activities under the direction of the Bureau of Medicine and Surgery.

Naval Aerospace Medical Institute, Naval Aerospace Medical Center, Pensacola, Fla.:

"Autonomic Responses to Vestibular Stimulation" by Pei Chin Tang and Bo E. Gernandt. Army-Navy Joint Report, NAMI Report No. NAMI-1066, April 11, 1969.

"Comparison of the Intraretinal b-Wave and d.c. Component in the Area Centralis of Cat Retina" by R. H. Steinberg. *Vision Research,* Vol. 9, 1969.

"Evaluation of Head Protection in Aircraft" by C. L. Ewing and A. Marshall Irving. *Aerospace Medicine,* Vol. 40, No. 6, June 1969.

"High-Intensity Effects on Slow Potentials and Ganglion Cell Activity in the Area Centralis of Cat Retina" by R. H. Steinberg. *Vision Research,* Vol. 9, 1969.

"Personality Characteristics of Jet Pilots as Measured by the Edwards Personal Preference Schedule" by G. E. Fry and R. F. Reinhardt. *Aerospace Medicine,* Vol. 40, No. 5, May 1969.

"Venting Alarm System for Cryogenic Liquids" by E. A. Molina and V. R. Reno. NASA-NAMI Joint Report, Report No. NAMI-1059, February 10, 1969.

Naval Medical Neuropsychiatric Research Unit, San Diego, Calif.:

"Fleet Effectiveness Prediction Studies at a Recruit Training Command" by John A. Plag and Jerry M. Goffman. *ONR Naval Research Reviews,* June 1968.

"Instrumentation for Sleep Research" by L. C. Johnson and Paul Naitch. *American Psychologist,* Vol. 24, No. 3, March 1, 1969.

"Spectral Analysis of the EEG of Dominant and Non-Dominant Alpha Subjects During Waking and Sleeping" by L. Johnson, A. Lubin, P. Naitch, C. Nute, and M. Austin. *Electroenceph Clin Neurophysiol,* Vol. 26, 1969.

Naval Hospital, Camp Pendleton, Calif.:

"Traumatic Intrahepatic Haemobilia Complicated by Terminal Rupture of the Aortic Valves" by F. E. Jackson, R. Burke, and W. Bramlett. *Far East Med J,* Vol. 4, December 1968.

Naval Medical Research Institute, National Naval Medical Center, Bethesda, Md.:

"Adrenergic Blood Pressure Responses in the Shark" by Sorell L. Schwarta and Joseph F. Borzelleca. *Science,* Vol. 163, January 24, 1969.

"ATPase Content of Striated Muscle Stressed in O_2/CO_2 and Hyperbaric $N_2/O_2/CO_2$ Atmospheres" by L. S. Friess and M. J. Cowan. *Toxicology and Applied Pharmacology,* Vol. 14, 1969.

"Changes in Lung Compliance in Experimental Hemorrhagic Shock and Resuscitation" by H. J. Proctor, G. S. Moss, L. D. Homer, and D. B. Litt. *Annals of Surgery* Vol. 169, No. 1, January 1969.

"Choicepoints in the Classification of Scientific Knowledge" by Irwin Altman. *People, Groups, and Organizations.* Columbia University, 1968.

"Comparative Fine Structure Study of the Gametocytes to Avian, Reptilian, and Mammalian Malarial Parasites" by Masamichi Aikawa, Clay G. Huff, and Helmuth Sprinz. *Journal of Ultrastructure Research,* Vol. 26, 1969.

"Effect of Solvent Viscosity on the Fluorescence of Tryptophan Derivatives" by Ira Weinryb. *Biochemical and Biophysical Research Communications,* Vol. 34, No. 6, 1969.

"Experimental Frostbite: Effect of 'Double Freeze' on Tissue Survival in the Mouse Foot" by E. Hardenbergh and R. Ramsbottom. *Cryobiology,* Vol. 5, No. 5, 1969.

"In Vivo Storage of Primate Kidneys" by E. L. Dupree, Jr., W. M. Abbott, and K. W. Sell. *The Lancet,* April 26, 1968.

"Multiplication and Differentiation of Trypansoma cruizi in an Insect Cell Culture System" by Duell E. Wood and Alan C. Pipkin, Sr. *Experimental Parasitology,* Vol. 24, No. 2, April 1969.

"Physiological Studies of the Mark IX Mixed Gas Scuba" by N. R. Antonisen. NMRI Research Report No. 1, May 1, 1969.

"Properties of Some Phenylalanyl Peptides in Ethylene Glycol-Aqueous Buffer Solvent" by Ira Weinryb and Robert F. Steiner. *Archives of Biochemistry and Biophysics,* Vol. 131, No. 1, April 1969.

"Role of Exogenous Adenosine Triphosphate in Catabolic and Synthetic Activities of Chlamydia psittaci" by Emilio Weiss and Noralee N. Wilson. *Journal of Bacteriology,* Vol. 97, No. 2, February 1969.

"Soybean Inhibitors. II. Preparative Electrophoretic Purification of Soybean Proteinase Inhibitors on Polyacrylamide Gel" by Victor Frattali and Robert F. Steiner. *Analytical Biochemistry,* Vol. 27, No. 2, February 1969.

"Specificity of the ^{3}H-Proline Incorporation Test as a Measure of Bone Matrix Formation" by Sandy C. Marks, Jr. *Biochemical and Biophysical Research Communications,* Vol. 35, No. 2, 1969.

"Studies of Bladder Stone Disease in Thailand" by Aree Valyasevi. Sakorn Dhanamitta, and Robert Van Reen. *American Journal of Clinical Nutrition,* Vol. 22, No. 2, February 1969.

"Tertiary Structure Determinants in Transfer RNA I. Pseudouridine" by David B. Millar. *Biochimica at Biophysica Acta,* Vol. 174, 1969.

Naval Submarine Medical Research Laboratory, Naval Submarine Center, Groton, Conn.:

"Effect of Rapid Eye Movement (Dreaming) Sleep

Deprivation on Retention of Avoidance Learning in Rats" by Chester A. Pearlman. SMC Report No. 563, February 3, 1969.

"Mammalian Adaptations to Diving" by Michael B. Strauss. SMC Report No. 562, January 28, 1969.

"Maximum Rate Kinetic Analysis Applied to Enzyme Assay Data" by Donald V. Tappan. SMC Report No. 564, February 5, 1969.

"Oral Acidogenic Bacteria in the Antarctic" by Peter Kasenchak and William R. Shiller, *Military Medicine,* Vol. 133, No. 1, January 1968.

DENTAL SECTION

PERSONNEL AND PROFESSIONAL NOTES

WAR MEMORIAL DEDICATION CEREMONIES

RADM Edward C. Raffetto, Assistant Chief of the Bureau of Medicine and Surgery (Dentistry) and Chief, Dental Division, delivered one of the messages at the dedication ceremonies of the American Dental Association War Memorial Court. A bronze sculpture was dedicated to the 160 dentists killed in wars during the twentieth century. Of that number, 30 were members of the Naval Dental Corps.

DENTAL OFFICER EDUCATION PROGRAMS—NAVMED P-5093

The 1969 revision of *Dental Officer Education Programs* has been distributed to all activities having dental officers assigned. This publication provides a current listing and comprehensive discussion of the various training opportunities available. The increasingly higher professional capability of dental officers may, basically, be attributed to the Naval Dental Corps' educational programs.

NAVY PERIODONTAL SCREENING EXAMINATION

The Naval Dental Corps is focusing attention on the control and prevention of periodontal disease. The Periodontal Screening Examination has many objectives but is specifically designed as an aid in the *early* detection and prevention of periodontal disease and in evaluating the effectiveness of treatment and oral hygiene procedures. Forms for the Navy Periodontal Disease Index, NAVMED 6600/1, and the Navy Plaque Index, NAVMED 6600/2, may be requisitioned through normal supply channels. The requisition numbers are S/N 0105-216-6610 and S/N 0105-216-6620 respectively.

PREVENTIVE DENTISTRY NAVMED P-5087

Chapter 5, *Prevention of Periodontal Disease,* has been distributed to all naval activities having dental personnel assigned, for inclusion in the preventive dentistry manual.

It is expected that all dental officers will be thoroughly familiar with the Five-Level Preventive Periodontics Program and will give maximum support to its implementation.

ANNUAL MEETING OF ASSOCIATION OF MILITARY SURGEONS

The annual convention of the Association of Military Surgeons of the United States will be held at the Sheraton Park Hotel, Washington, D.C., November 16-19, 1969. The Dental Section will meet on Monday afternoon, November 17, from 1300-1600.

The theme of the Dental Section Program is "Dental Research and Development Related to Clinical Dentistry." CDR J. P. Kelly, DC USN, Chief of Oral Surgery, Naval Hospital, Chelsea, Massachusetts, will speak on the subject "Rehabilitation of Maxillofacial Battle Casualties—1969."

ILLEGAL OR IMPROPER USE OF DRUGS/NARCOTICS/MARIJUANA

SECNAVINST 6710.1A of 11 July 1969 requires commanders to institute and conduct a rigorous program to counteract the illegal experimentation with and use of marijuana, narcotics, and dangerous drugs by naval personnel, and specifies reports to be rendered.

Enclosures to the instruction include a comprehensive list of the restricted habit-forming drugs as well as information concerning the dangers of LSD and marijuana usage, and a list of Navy motion picture films and printed matter currently available for instructional purposes.

PROFESSIONAL RELATIONS PROGRAM

NAVAL DENTAL CORPS PARTICIPATION ANNUAL SESSION OF THE ADA

The new Joint Armed Forces Dental Exhibit Military Contributions to Dentistry will be shown at the 110th Annual Session of the American Dental Association, October 12-16, 1969, at New York City.

Two officers will appear on the program as panelists in operative dentistry. CAPT Theodore R. Hunley, DC USN, will present "Pin Placement Technic" and CDR James D. Enoch, DC USN, will present "Rationale for Pin Utilization."

CAPT Gordon H. Rovelstad, DC USN, will present a paper, "The Application of Dental Research in the Armed Forces Dental Services" at the International Conference on Military Dentistry planned by the Commission on Armed Forces Dental Services of the Federation Dentaire Internationale.

Ten officers of the Naval Dental Corps will attend meetings of the various Councils of the American Dental Association.

ARMY RESERVE UNIT VISITS NAVAL DENTAL SCHOOL

On July 31, the Army's 338th Dental Detachment (KJ) from the Boston Army Base toured the Naval Dental School, National Naval Medical Center, Bethesda, Maryland. Led by Commanding Officer Lieutenant Colonel Stanley H. Short, DC USAR, the 15 dental officers and 20 enlisted men were welcomed to the Dental School by CAPT Howard B. Marble, Jr., DC USN, Head of the Oral Surgery Department, who then conducted the group through the School's facilities.

Composed of Reserve personnel actively engaged in civilian dental practice, the 338th Detachment is a self-sustaining dental unit with capabilities in oral surgery, prosthetic treatment and general dentistry.

The unit which was stationed at Fort George E. Meade for 2 weeks active duty also toured Walter Reed Army Medical Center, Washington, D.C.

RESERVE DENTAL OFFICERS SERVE AS A UNIT

Five Naval Reserve dentists, part of Naval Reserve Dental Company 3-4 from Huntington, New York, recently served on active duty as a unit.

The officers were assigned to the Marine Corps Recruit Depot, Parris Island, South Carolina for the fourteen day period.

Members of the team were CDR Charles F. Shreier, DC USNR-R, CDR Edward J. O'Shea, DC USNR-R, LCDR Michael W. Diamond, DC USNR-R, LCDR John C. Vogeley, DC USNR-R, and LCDR George C. O'Malley, DC USNR-R.

ARTICLES AND ABSTRACTS

EFFECT OF CHEMICAL AGENTS ON PROGRESS OF CUTANEOUS WOUND HEALING

LCDR J. E. Trainor, DC USN and LCDR C. A. Brown, DC USN.

In order to accurately assess the claims made for the effect of therapeutic agents on wound repair, firmly established standards for measuring the histologic changes that occur in normal wound healing should be used. The purpose of this study was to determine, by applying previously suggested standards for measuring the progress of healing, the effect of commonly used antiseptic solutions on the overall rate of healing. Standardized cutaneous incisions were made on the backs of three Minnesota Miniature pigs at various intervals of time from 1 to 21 days. Each of four series of wounds was treated with a different antiseptic agent, and two series were left untreated to constitute controls. Microscopic sections were prepared from block biopsies, and the normal healing process was compared histologically with healing in wounds treated with an antiseptic. Three major recovery phases were noted. Phase I occurred within

the first 3 days postoperatively and consisted of epithelial inversion, epithelial union, and reticular fiber concentration. Phase II occurred from the fourth to the seventh day and involved fibroblastic proliferation, angioblastic proliferation, and increased deposition of collagen. Phase III occurred after the seventh day and included organization of the new fibrous connective tissue, horizontal orientation of the new fibers, and decreased cellularity within the wound. The treated wounds showed varying delays in the first two phases of healing but no prolongation of the overall healing process. An emulsion containing hexachlorophene (Phisohex) produced a slight delay in the completion of phase II. Povidone-iodine (Betadine), iodine, and thimerosal (Merthiolate) produced delays in the completion of both phase I and phase II. After 10 days there was no noticeable histologic difference between the controls and the treated wounds. It was concluded that although healing was delayed in the early phases of repair, in the wounds treated with antiseptics the overall rate of healing was not affected by the agents used in this study.

(Abstract by Research Work Unit: MR005.19-6052 by LCDR J. E. Trainor, DC USN and LCDR C. A. Brown, DC USN.)

The opinions and assertions contained herein are those of the authors and are not to be construed as reflecting the views of the Navy Department or the naval service at large.

EVALUATION OF A COMMERCIALLY PROPORTIONED, MECHANICALLY MIXED SILICATE CEMENT

LCDR R. R. Eklind, DC USN and LCDR D. A. Hansen, DC USN.

Some studies indicate that the physical properties of mechanically mixed silicate cements surpass the results of hand spatulation. However, the problem of accurately measuring small quantities of the ingredients to ensure a standard powder-liquid ratio has prevented mechanical mixing of silicate cements from gaining wide clinical acceptance. A commercially prepared preproportioned capsule of silicate cement powder and liquid (Silicap) is now available for use with a special mechanical mixer (Silamat). The purpose of this study was to evaluate the cement prepared from the mechanically mixed capsules and to compare it with hand spatulated silicate cement. The degree of uniformity in quantity of powder and liquid was determined by weighing to the nearest milligram the materials in 10 capsules. Consistency tests and setting time determinations were carried out according to A.D.A. specification #9 on cement prepared from another 10 capsules mixed mechanically. A number of mixes of hand spatulated silicate cement (S.S. White) prepared by dentists or dental assistants were used for comparison. The quantity of material in each capsule was found to be 181 ± 1 mg of powder and 86 ± 1 mg of liquid. It was determined that 81 ± 1 mg of the liquid was actually mixed with the powder, providing a final powder-liquid ratio of 2.2 to 1. The amount of acid in the liquid was ascertained by titration with standard alkali and found to be the same in all samples. Setting time and consistency tests indicated no essential difference between the preproportioned, mechanically mixed material and the hand spatulated samples. Certain advantages of using the capsule and mechanical mixer combination were noted, such as uniformity of product, ease of handling of materials, and the possibility of using the mixer for amalgam.

(Abstract by Research Work Unit: MR005. 19-6052 by LCDR R. R. Eklind, DC USN and LCDR D. A. Hansen, DC USN.)

The opinions and assertions contained herein are those of the authors and are not to be construed as reflecting the views of the Navy Department or the naval service at large.

PREVALENCE, INCIDENCE AND GEOGRAPHIC DISTRIBUTION OF CARIES-FREE NAVAL RECRUITS

CDR H. J. Keene, DC USN; CAPT G. H. Rovelstad, DC USN; CAPT S. Hoffman, DC USN; and CDR W. R. Shiller, DC USN.

A 9-year survey on 500,000 Great Lakes naval recruits indicated that only 2 men per 1,000 (0.2 percent) had no previous history of dental caries experience at the time of entrance into the Navy. The annual incidence of caries-free men ranged from a low of 1.3 per 1,000 in 1967 to a high of 3.1 per 1,000 in 1963. Marked variations in the prevalence of caries-free men from different geographic regions of the United States were observed. Correlation analysis indicated that these variations were partly related to the availability of natural fluoride in public water supplies.

(Abstracted by Research Work Unit: NDRI PR 69-08 by CDR H. J. Keene, DC USN; CAPT G. H.

Rovelstad, DC USN; CAPT S. Hoffman, DC USN; and CDR W. R. Shiller, DC USN.)

The opinions and assertions contained herein are those of the authors and are not to be construed as reflecting the views of the Navy Department or the naval service at large.

BACTERIAL AEROSOLS GENERATED DURING DENTAL PROCEDURES

Dent Abs 14(6):357–358, June 1969.

Some dental procedures generate aerosols with bacterial concentrations that exceed those produced during coughing or sneezing.

A controlled environment operatory and human aerosol test chamber were used to study characteristics of bacterial aerosols generated from a patient's mouth during dental procedures and by common naso-oral activities. The test chamber (a 30x30x90-cm rectangular stainless steel box with tapered ends) was suspended above a dental chair. The top, fitted with a 20x30-cm window, and the sides with glove ports, sleeves, and surgeon's gloves, allowed the dentist to see and operate. A manifold attached to the front of the chamber had outlets for four air samples and a bypass valve.

Dental procedures such as oral examination and use of hand instruments produced aerosols with bacterial concentrations of about first-order magnitude and were equivalent to aerosols produced when a patient speaks or breathes.

A prophylaxis handpiece used with a pumice cup and pumice to clean teeth, an air turbine handpiece with air coolant, and air spray from a three-way syringe produced numbers of bacteria comparable to those resulting from coughing.

An air turbine handpiece, when used with air-water spray coolant, atomized 20 times greater numbers of bacteria than with air spray alone. This concentration was numerically equivalent to the aerosols produced during most oral activities, including sneezing. The air-water sprays also produced aerosols with the greatest percentage of particles 5um or less in diameter.

The rotary action of the bristle disk during polishing procedures and the use of the air-water spray from the three-way syringe produced bacterial aerosols equal to or exceeding those produced during all oral activities studied, including those considered unsanitary when at close range.

The data demonstrate that a reduction of almost three orders of magnitude in bacterial aerosols escaping from the mouth can be effected by using high-velocity air suction during dental procedures. To be effective, the suction tip must be maintained well inside the mouth and must remain unobstructed during the entire course of the dental procedure.

As there is presumptive evidence that a potential health hazard exists, it is only reasonable that the dentist and auxiliary personnel should not permit repeated exposure to bacterial aerosols with concentration equal to or exceeding those produced by naso-oral activities considered unsanitary for the intimate space occupied.

(Micik, Rudolph E.; Miller, Robert L; Mazzarella, Maurice A; and Ryge, Gunnar. USPHS Dental Health Center, San Francisco, Calif. Studies on dental aerobiology: I. Bacterial aerosols generated during dental procedures. J Dent Res 48: 49–56, Jan–Feb 1969.)

ODYSSEY OF A PROFESSION

Seymour J. Kreshover, J Balti Coll Dent Surg 23(a):39–44, Dec 1968.

The many changes occurring today impose special obligations upon our profession and provide opportunities to better serve mankind. People today believe health care is a right rather than a privilege and the future demands for health services will be unprecedented. If we are to meet this increased demand, we must be responsive to change.

It is important to note a few specific trends. Not only are trade unions seeking broader health benefits, but more health insurance covering dentistry is expected, and expanded Medicare programs will have a marked impact on dental care. Dentistry will also become involved in more "total" health care programs.

As a result of these forces, the demand for dental care will increase by 50–75 percent by 1975. Dentists will have to judiciously transfer selected duties to auxiliaries, as will the whole health world. To meet the public's desire for unfragmented care, dental education and practice will be more hospital oriented. Continuing education will assure the high quality services the profession is obliged to render.

Dentistry, the university, and the Federal Government are becoming partners in providing what society demands. It is not enough to train professional personnel—there must also be a balanced program

of education, service, and research, as well as concern for disease prevention. To help meet these responsibilities new programs of Federal support are being explored.

The Federal investment in health is $15.6 billion, three times more than five years ago, of which $1.2 billion is currently spent by the National Institutes of Health for biomedical research and training.

At the National Institute of Dental Research, two current research areas are caries and the development of restorative materials. The role of certain streptococci in initiating caries has been shown and investigators have recently isolated the enzyme dextranase, which dissolves dextran—the gummy substance that forms the matrix of the bacterial plaque. Pending the eradication of caries, the development of a truly adhesive restorative material is a practical necessity. Improved biomaterials are also needed for treating victims of oral-facial diseases and trauma. Basic research on periodontal disease and other oral disorders will hopefully lead to better management and ultimate prevention.

The odyssey of dentistry today is of greater dimensions and complexity than our founding fathers may have ever foreseen.

(Abstracted by CDR H. C. Pebley, DC USN.)

NURSE CORPS SECTION

CORONARY CARE UNIT NURSING

The following article was written by LCDR Ann P. Connors, NC, USN who is presently assigned to the Naval Hospital, Chelsea, Mass.

"Possibly 100,000 patients with hearts that are too good to die can be saved each year with proper surveillance and treatment in acute coronary care units." This statement, by an eminent cardiologist, points out the dramatic mortality reduction achievable in hospitals through continuous monitoring of coronary patients during the critical stages of their disease. Experience since 1962, when the first coronary care units opened at Bethany Hospital in Kansas City and Presbyterian Hospital in Philadelphia, has shown that mortality rates for acute myocardial infarction in these institutions have been reduced 50 to 60 percent.

Seventy-five percent of all patients with myocardial infarction exhibit significant arrhythmias during the course of their illness. Experience in coronary care units indicates that the sooner these arrhythmias are recognized and proper treatment begun, the lower the mortality rate and incidence of progression into more severe forms. Further reduction in the mortality rate is expected as greater knowledge concerning the genesis and significance of specific arrhythmias is gained.

Anticipating the opening of a two to four bed coronary care unit, I was afforded the opportunity to attend a four week course in coronary care unit nursing. The intensive program was held under the auspices of the U.S. Department of Public Health with Boston University as the cooperating professional facility. All classes were conducted at Boston University Hospital, Boston, Massachusetts.

The student enrollment was limited to nineteen nurses selected from hospitals as far south as Kentucky and westward to Michigan, all with varying degrees of experience in coronary care unit nursing.

The classes consisted of lectures, laboratory participation, self-study groups and clinical experience in two participating hospitals with established coronary care units. The faculty consisted of three instructors, guest lecturers which included physicians, physiologists, psychiatrists, nurses, dieticians, social workers, public health nurses, electrical engineer and a former patient of a coronary care unit.

The curriculum was composed of pertinent anatomy and physiology, homeostasis in fluid and electrolyte balance, electrocardiographic interpretation of cardio-arrhythmias, techniques of electrocardiography, medical electronics, pharmacodynamics of cardiovascular drugs, pathophysiology, cardiac electrophysiology, cardiopulmonary resuscitation and defibrillation techniques, coronary care unit administration and policies, legal aspects of coronary care unit nursing and public health aspects of coronary disease.

The first two weeks' study were heavily concentrated in the areas of cardiology especially in arrhythmia recognition and emergency treatment. The

roles and responsibilities of the coronary care unit nurse were stressed during this period as were the philosophy and goals of the unit itself. The last two weeks of the course were devoted to more technical aspects of coronary care including emergency resuscitation measures, dog laboratory observation and defibrillation practice sessions, EKG taking, EKG tracing sessions with a cardiologist, Resusci-Anne practice, and finally our clinical field experience at the coronary care units of Boston University Medical Center and the Carney Hospital in Dorchester, Mass.

Eighty-five percent of the deaths from myocardial infarction occur within the first week of the attack. The vast majority of these deaths result from cardiac arrhythmia, cardiogenic shock, and cardiac failure, alone or in combination. Rapidly accumulating experience reveals that prompt and effective treatment in the hospital will significantly reduce this mortality, particularly if the potentially lethal cardiac arrhythmias (ventricular fibrillation, ventricular tachycardia and asystole) are promptly detected, diagnosed and treated by the application of modern techniques in the hands of a competent staff. This requires continual attention and instantaneous action. It is, therefore, the mission of a coronary care unit to provide patients with known or suspected myocardial infarction constant and intensive care, to facilitate early detection of complications, and provide prompt, effective treatment.

It should be emphasized that admission to a special unit is based on the established or suspected diagnosis of myocardial infarction, and not the clinical state of the patient. In other words, a patient with probable acute myocardial infarction should be admitted even though, at the time of arrival, he is in no distress and appears physiologically stable. The usual complications, arrhythmia, shock and congestive heart failure, frequently occur transiently and unpredictably; in addition, minimal delay in therapy is required to ensure success. Complications are associated with a poor prognosis but nevertheless often occur in patients with an apparently salvageable myocardium. Thus, all acute coronary patients are candidiates for cardiopulmonary emergencies and ideally should be placed in an environment where prompt detection and treatment may be realistically anticipated.

Several elements are viewed as essential to the successful functioning of coronary care units. Especially important is the development of a cadre of well-trained nurses, educated in the care of the coronary patient. They should be trained in the proper utilization of available electronic and mechanical devices and motivated not only to care for a group of patients with guarded prognosis but to take the initiative in time of emergency. Each patient's electrocardiogram is constantly monitored at his bedside and at the nursing station. An automatic alarm signal is set to indicate any violation of acceptable cardiac rate and rhythm. Special supportive equipment for defibrillation, heart activation, cardiopulmonary resuscitation and emergency drug therapy must also be immediately available.

The short-term or long-term prognosis of acute myocardial infarction is influenced by a host of factors, including pre-existing cardiac or systemic pathology, extent of infarction, complications, and the current status of medical and nursing care. There is much literature and evidence to suggest that the potential lethality of complications is substantially reduced or reversed when superior therapy is afforded, thereby supporting the concept that the special care elements contained in a coronary care unit will predictably reduce fatality rates. It is of particular importance to recognize that sudden death in acute myocardial infarction is not necessarily related to the extent of the infarction but may be the result of some immediate intervening transient event. If the insult is successfully treated, satisfactory cardiac function may be resumed. These are the hearts "too good to die." Reports of two series indicate that from 50 to 70 percent of the deaths from acute myocardial infarction in the first 24 to 48 hours after onset do not present discernible causes of death at autopsy. It is assumed that these fatalities resulted from "electrical causes," VIZ., ventricular fibrillation or standstill. It is reasonable to assume that under ideal conditions today many of these could be successfully resuscitated.

One form of cardiac arrhythmia, ventricular fibrillation, accounts for from 35 to 50 percent of the early deaths after infarction. It is probably 100 percent fatal unless successfully treated. Cardiac arrest, ventricular standstill, may well occur with equal frequency. Both conditions present a functional state of "cardiac arrest" without effective pumping action of the heart and both conditions are uniformly fatal unless effective cardiac action is promptly restored. Ventricular tachycardia is commonly observed and is hazardous because it may evolve into ventricular fibrillation and, as with all rapid arrhythmias, the rapid heart rate itself compromises the integrity of the circulation. Sustained

rapid arrhythmias of any cause may lead to congestive heart failure.

Modern developments in the treatment of cardiac arrest (ventricular fibrillation or standstill) and the other important arrhythmias have greatly increased the chances of the patient for survival. External electrical countershock, closed-chest cardiorespiratory resuscitation, improved ventilatory techniques, and advances in drug therapy serve to counteract complications proven lethal if untreated. Arrhythmias such as ventricular tachycardia, which carry a high fatality rate, can now be treated with a high degree of success. However, the *maximal benefit* from these therapeutic and supportive measures is contingent on *early recognition* and *prompt action*. The inherent services provided by coronary care units assure superior clinical treatment for arrhythmias and other major complications as well.

Usually, the low patient-nurse ratio necessary for constant surveillance and care in such a unit provides an opportunity for the staff to know patients as individuals. Nursing care that strives to meet individual physiological, emotional and social needs can be achieved if the goal for such care is established. The electronic monitoring equipment is but one of the many tools that the nursing staff uses to maintain competent observation. Another tool is knowledge—knowledge of the disease, of medical and nursing measures employed for treatment and prevention of complications, of the patterns of psychological reaction to sudden serious illness, the various adjustments which confront patients and the significance of the family in all stages of a patient's recovery. A third tool is communication—with the patient, the physician, the family and other professional staff members. It is only through the team effort of all persons involved that the ideal of patient-centered care can be achieved. Therefore, with this goal in mind, we hope to introduce a series of in-service educational programs aimed toward consideration of the basic arrhythmias and emergency nursing measures used in coronary care nursing. It is important that the staff working on other wards, to which patients are later transferred, be familiar with the coronary care unit in order to assist the patient in his continued adjustment after transfer, thereby sharing in the satisfaction of nursing a "heart too good to die."

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PREVENTIVE MEDICINE SECTION

LOUSE-BORNE RELAPSING FEVER

WHO Wkly Epid Rec 44(26): 425–426, June 27, 1969.

Louse-borne relapsing fever is becoming less and less important, particularly with respect to the risk of dissemination of this disease through international traffic. In the past the disease has occurred in sporadic outbreaks which sometimes with great rapidity spread across whole continents, thereafter to die down and disappear often for long periods of time.

A serious epidemic occurred from 1942–1944 which began in North Africa and spread to the Eastern Mediterranean and Europe. An estimated 1 million persons became ill, of whom 50,000 died. Since then, louse-borne relapsing fever has mainly occurred in a few relatively small areas of Africa.

The disease occurs where overcrowded populations live under unhygienic conditions favoring the wide dissemination of human body lice. Insecticides make it possible to check the spread of the disease through rapid control of the vector.

Reporting of this disease is not always very reliable because it may occur concomitantly with louse-borne typhus fever and indeed with the tick-borne variety of relapsing fever. At present, countries do not always indicate which type of relapsing fever they are reporting. Some health administrations only report cases admitted to hospital, while others main-

tain more complete reporting, sometimes with laboratory confirmation of diagnosis.

Ethiopia is the only country which has continuously reported a high number of cases of louse-borne relapsing fever since 1950. Ethiopia reported approximately 4,700 cases annually during the fifties and about 4,300 cases during the first 8 years of the sixties. However, it is noteworthy that during the period 1950–1967 there was a considerable variation from year to year in the number of cases reported, from some 2,300 cases in 1951 to some 8,800 cases in 1959. Cambodia, with some 4,200 cases, was responsible for 56% of the louse-borne relapsing fever reported in 1950 but has not reported a single case during the last 10 years.

Foci of louse-borne relapsing fever of considerable importance exist in the Ethiopian highlands. In 1968, louse-borne relapsing fever was reported from all provinces in Ethiopia and 5 provinces in the Sudan (Blue Nile, Upper Nile, Kassala, Khartoum and Kordofan). Some of the latter border on the more heavily infected Ethiopian provinces, a fact which in addition to the history of relapsing fever in the Sudan, stresses the urgent need for the development and maintenance of an effective surveillance system for this disease.

Since 1950 the Sudan has reported less than 100 cases annually. There were years when no cases were reported at all, until 1968 when close to 2,000 cases occurred.

Although the increase in the number of cases reported in 1968 is considerable, the outbreaks are confined to a limited area of Africa.

TETANUS

USDHEW PHS NCDC Tetanus Surveil Rep No. 2, 1–2, Apr 1969.

Data derived from 234 cases of tetanus occurring in the United States during 1967 confirm the major conclusions expressed in the first report of nationwide tetanus surveillance. The disease is increasingly one of the older segments of the U.S. population, with half the cases 54 years of age or older. The national case fatality ratio was 67%, 76% in neonates and 80% in persons 50 years or older. The disease was 5 times more common in nonwhites than whites, and males outnumbered females by 3:2. The southernmost tier of states continue to lead the nation in incidence and more cases occurred in the spring and summer months than in winter.

Injuries occurring at home or in the garden accounted for over 70% of tetanus predisposing wounds. A long incubation period of 10 days or more was a reliable index of significantly better prognosis only in patients less than 50 years old. Generalized convulsions were associated with significantly poorer prognosis in patients less than 20 years of age and 50 years or older.

There were no significant differences between groups which received various forms of antitoxin therapy and no serotherapy, although lower case fatality ratios were seen in antitoxin treated groups. Treatment groups were relatively small and the use of hyperimmune human globulin appears to be supplanting animal antitoxin.

The need for universal immunization against tetanus is clear.

For 1967, 263 cases of tetanus were officially reported from 30 states to the NCDC, Atlanta, Georgia, through the National Morbidity Reporting System (NMRS). Thirty-nine cases were reported from the Commonwealth of Puerto Rico. Following the initial telegraphic notification of cases by the NMRS, 234 tetanus surveillance forms were received by NCDC for cases occurring in the United States and 32 from Puerto Rico.

The national incidence of tetanus for 1967 was 0.12 per 100,000 population, which was unchanged from 1965 and 1966.

The incidence of neonatal tetanus (10% of all cases) was 0.233 per 100,000 live births for whites and 2.94 per 100,000 nonwhite births.

Despite modern therapeutic innovations, the overall case fatality ratio from tetanus has not significantly changed since 1950.

PLAGUE—1968

USDHEW PHS NCDC Morb & Mort Wkly Rep 18(30):262, July 26, 1969.

During 1968 the total number of plague cases for the world reported to the World Health Organization was 1,318 cases with 160 deaths. Of the total cases, 973 with 90 deaths occurred in Asia with Vietnam contributing 780 cases and 37 deaths. The total for the world exclusive of the Vietnam cases continued to decline from the annual totals for recent years.

The recorded number of cases in the world reached its lowest point in 1959. Until that time India, Indonesia, and Burma were contributing the majority of

plague cases to the world total, although the disease was steadily declining in all 3 countries. Since then, the total number of reported cases has increased due to the increasing number of cases in Vietnam. During 1963–66 South America was contributing most of the world's total until Asia reflecting the increase in Vietnam, again reported more cases.

The number of cases in 1968 approached the level of 15 years ago, but the distribution by country has changed. India, once the most affected country, reported no cases in 1968 for the first time in its recent history. In Indonesia, where the disease seemed to disappear in 1959, it reappeared in 1968 in the same district and in the same locality where it had last been reported. In Nepal, there were 13 cases and 12 deaths. Thus plague in Asia not only increased in number of cases but also occurred in more territories than in recent years. No large outbreaks occurred in Africa or in the Americas; however, countries in these areas reported plague cases similar in number to previous years.

As in every year in the last decade, most of the cases in 1968 were bubonic although pulmonary plague did occur.

MELIOIDOSIS

USDHEW PHS NCDC Morb & Mort Wkly Rep 18(32):278–279, Aug 9, 1969.

Melioidosis was recently diagnosed in a male rhesus monkey (*Macaca mulatta*) being used in psychological research at the National Institutes of Health (NIH). The monkey was received at NIH on 1 Oct 1968, in a shipment of 50 from India. His only overt illness occurred on 7 Oct 1968, when he had soft stools for which he received tetracycline, nitrofurazone, and a commercially-made oral feeding mixture. On 2 Dec 1968 he was issued to a psychology laboratory, where on 18 Dec he underwent a craniotomy with excision of parts of the cortical sensory areas. Sensory testing was started on 15 Jan 1969, but the monkey proved difficult to test and train.

In late April 1969 round scabs were noted at the surgical scar; by mid-May they appeared raised and were thought to be underlaid by abscesses. On 19 May the lesions were distinctly suppurative, and at this time similar processes were noticed on the skin of the chest and leg. The superficial head and chest lesions were cultured. The former site yielded a mixed flora of *Staphylococcus aureus, Enterobacteriaceae,* and *Proteus sp.* with a few colonies that were later shown to be *Pseudomonas pseudomallei.* Culture of the chest lesion yielded predominantly *P. pseudomallei.*

The animal was sacrificed on 28 May. Blood values at that time were: hematocrit 32%; hemoglobin 9.1 g/100 ml; RBC 4,490,000; and WBC 24,850, with 84.5% neutrophils, 15% lymphocytes, and 0.5% monocytes. At necropsy a 2 cm. raised, fluctuant, subcutaneous abscess was observed on the left dorsal aspect of the head, directly over the site of the previous frontoparietal craniotomy. The abscess contained thick, pale, yellow pus. A similar subcutaneous abscess was located on the left chest, approximately 1 cm. lateral to the nipple, and was connected by a fistulous tract to a larger 3 by 4 cm. abscess in the left axilla, apparently involving the axillary lymph nodes. Internally, multiple 0.5 to 1 cm. abscesses were observed in the liver, spleen, pancreatico-splenic lymph nodes, and in the superior gastric nodes. One of the liver abscesses was contiguous with the wall of the gallbladder. Two 1 cm. subpleural abscesses occurred in the dorsal aspect of the right apical lung lobe and lesions were found also in several mediastinal lymph nodes. The pus in the internal lesions was thin, dull white, and in some lesions appeared tinted pale green. Cultures taken from the head, chest, liver, and spleen yielded pure growth of *P. pseudomallei,* while culture of heart blood was negative. Identification of the organism was confirmed at the Walter Reed Army Institute of Research and at NCDC.

Editorial Comment: This report represents the 4th culture positive case of melioidosis in imported nonhuman primates reported this year. Melioidosis was previously diagnosed in 2 stump-tailed macaques and a chimpanzee. The first reported case had a history of a chronically discharging lesion present at the time of importation. The next 2 cases first showed signs of disease at the site of implanted foreign objects. The current case first showed signs of disease at the site of an old surgical wound. A serological survey currently in progress indicates that a significant number of monkeys from Southeast Asia have titers to *P. pseudomallei.*

GROUP A MENINGOCOCCAL MENINGITIS —BOSTON, MASSACHUSETTS

USDHEW PHS NCDC Morb & Mort Wkly Rep 18(23):197–198, June 7, 1969.

Since the first report in 1967 of a case of Group A meningococcal infection to NCDC, Atlanta, Geor-

gia, 2 cases have been reported from Boston City Hospital during the week of 30 May 1969. One case, a 6-year-old boy was hospitalized on 24 May with a 24-hour history of vomiting, progressive irritability, and neck pain. Physical examination revealed a febrile (101.4° F.), normotensive, semicomatose boy with nuchal rigidity and generalized petechiae. A lumbar puncture revealed cloudy cerebrospinal fluid and gram-negative intracellular diplococci were seen on microscopic examination. Intravenous penicillin was given and within 48 hours, the boy was afebrile and alert. Group A *Neisseria meningitidis* was isolated from 2 blood cultures as well as the cerebrospinal fluid.

The other case was a 50-year-old man admitted on 27 May with a 16-hour history of headache and progressive obtundation. On physical examination, the patient was febrile (100.6° F.) but normotensive and no petechiae were seen. He was semicomatose and combative with marked nuchal rigidity. Lumbar puncture revealed turbid cerebrospinal fluid with a cell count of 8,700, all polymorphonuclear leukocytes on smear. Gram-negative intracellular diplococci were seen on smear of the cerebrospinal fluid, and intravenous penicillin was begun. Despite a series of grand mal seizures shortly after admission, the patient rapidly improved and was alert and afebrile 36 hours after admission. Group *A N. meningitidis* was isolated from the cerebrospinal fluid.

Both meningococcal isolates were sensitive to sufadiazine being inhibited at/or below concentrations of 0.78 μg per ml. (.078 mg%).

No connection could be established between these 2 persons, and no further cases of Group A meningococcal infection have occurred to date at Boston City Hospital.

CANINE SCABIES IN CHILDREN —"PUPPY DOG" DERMATITIS

Co. Los Angeles Hlth Dept Morb & Mort, May 10, 1969, Amer J Dis Child 117, 239–242, Feb 1969.

A widespread pruritic skin reaction in a child often poses a difficult diagnostic problem. Canine scabies can be responsible for such a widespread eruption. An affirmative answer to the question, "Is there a new puppy in the house?" often will be the physician's key to the diagnosis. If the puppy is infested with the mite *Sarcoptes scabiei,* variety *canis,* the intimate association between the child and the puppy leads to the transfer of organisms to the child. Even though this tiny mite prefers the dog as a host, it will not hesitate to burrow into the skin and secure a meal from a human.

The child with canine scabies is usually brought to the physician because of severe pruritus and a "rash" of several days' duration. The onset is usually sudden with the development of many lesions. The child is otherwise well. A papular or papulovesicular eruption is presented. Individual lesions, of which there can be hundreds, are usually small red papules 1 to 3 mm. in diameter with some assuming a vesicular appearance. There is a predilection for the lower chest, abdomen, and forearms. Scratching leads to much excoriation.

Occasionally adult animals have been found to be the source of the mites rather than a puppy. Fondling of puppies provides greater opportunity for transfer of organisms. A child often has related that he carried his puppy in his arms, close to the chest-abdomen area. On several occasions a child has remarked "the puppy was cold and I kept him warm by holding him inside my shirt." Diagnosis is aided not only by the history of a new puppy but also by finding several others in the family complaining of a similar condition. The degree of involvement may vary considerably among members of the family.

Further questioning regarding the dog's fur is important. Canine scabies, commonly called "Mange", causes patchy loss of hair with scaling in the dog. Often the animal, if greatly infested, will appear ill. The dog may have been taken to a veterinarian for confirmation of the diagnosis, by finding the *S. scabiei* in scrapings of scales taken from the involved areas. The scrapings are placed on a glass slide with a drop of immersion oil, covered with a cover slip, and examined for the organism with the low power objective of the microscope. The mite is less than 0.5 mm. in size.

There is a question as to whether or not the development of lesions in the patient with canine scabies is based on an allergic hypersensitivity reaction. If it is on an allergic basis, it would require a period of at least 3 to 4 days for the development of a hypersensitive state toward the antigenic material. In most patients, there is a period of exposure at least this long with no eruption. Also supporting the sensitization theory is the suddenness of onset with multiple lesions seen in many patients. This suggests introduction of antigenic material into various skin sites, without evident reaction prior to

sensitization. After sensitization occurs, there is a sudden development of lesions due to the presence of the antigenic material which had previously caused no reaction. The sensitization theory has been disputed because some patients react within hours after exposure; it may be that these patients had been sensitized in the past.

The mite of canine scabies appears to be morphologically similar to the mite of human scabies. There is a difference in the distribution pattern of lesions caused by the 2 types of mite. In human scabies the pruritic papular lesions tend to be located in the webs of the fingers, flexures of the arms, axillae, and area around the nipples, genitals, and buttocks. The course of the 2 conditions is also different. In the person infested with human scabies, the condition may persist for years. If the patient with canine scabies is removed from the source of mites, the involvement generally clears within a short period.

Clearing of the skin eruption following removal of the dog has led to the idea that the canine scabies mite feeds but does not propagate itself in the human skin. In the case of a patient whose eruption persisted, with new lesions forming for weeks after the dog had been removed from the environment, examination of scrapings revealed mite eggs. While on the warm microscope stage, movement developed in one egg which later hatched. Even though this organism does not seem to naturally select the human, it in fact is able to propagate itself in the human host.

Removal of the source of mites by recognition and treatment of the infested animal is the prime objective of care. This is best accomplished by a veterinarian. However, animals with widespread involvement are often difficult to cure. Destruction of the mites of the child can be accomplished by the use of topically applied crotamiton lotion (Eurax lotion) or gamma benzene hexachloride lotion (Kwell). The patient is instructed to take a warm bath with thorough washing. The entire body from the neck down is then anointed with the lotion. This is left in place for 24 hours, after which time another bath is taken. Clean clothing is then put on. The crotamiton lotion, besides being a scabicide, also has antipruritic properties which make its use most helpful.

Summary

A papular dermatitis can be caused by dog scabies. Typically it is located on the chest and abdominal areas. It is of rather sudden onset and often is noted in several members of the same family. A newly acquired puppy suffering from animal scabies (mange) can be the key to the diagnosis. A patient was examined in whose skin were found both mites and mite eggs. This observation indicates that propagation of dog scabies can occur in the human.

BABESIOSIS

USDHEW PHS NCDC Morb & Mort Wkly Rep 18(32): 277–278, Aug 9, 1969.

On 13 July 1969, a 59-year-old widow was admitted to a New Jersey hospital with a 2-week history of fever, headache, malaise, and weakness. A peripheral blood smear contained numerous atypical ring-like structures within the red blood cells, which resembled *Plasmodium falciparum* trophozoites. The patient gave no history of exposure to malaria, blood transfusions, or drug abuse, and medical evaluation uncovered no chronic or debilitating diseases. A normal splenic shadow was present on a barium enema examination.

For the past 20 years, the patient's travel had been limited to the continental United States and Hawaii. On 5 May, she and her 2-year-old dachshund left southern California and went to their summer home on Nantucket Island, Massachusetts. While there, the dog often chases, kills, and returns small rodents to the house. Because ticks are common on the island, the patient examined the dachshund daily and removed a number of ticks with tweezers or her fingers. In mid-May she removed a tick deeply embedded in her own suprasternal notch.

The patient was treated with chloroquine and gradually recovered, and the parasitemia disappeared. The pretreatment smears were reviewed at the National Malarial Repository, NCDC, and babesiosis was tentatively diagnosed on the basis of the morphology of the parasite and the absence of malarial pigment or circulating gametocytes.

The patient's blood was inoculated into various laboratory animals, and infection was established in a hamster. The appearance of the organism in human and hamster red blood cells is compatible with a rodent *Babesia* species, possibly *B. rodhaini.*

Editorial Note: Babesiosis is a cosmopolitan, tick-transmitted protozoal infection in many wild and domestic animals. In some animals it causes a febrile, hemolytic disease. The three human cases reported in the literature occurred in splenectomized individuals. This woman is the first apparently healthy individual in whom the disease has been recognized.

OCCURRENCE OF *SPOROTRICHUM SCHENCKII* IN COLD-STORED MEAT PRODUCT

USDHEW PHS CDC Veter Public Hlth Notes, P. 6, May 1969.

In the American Journal of Epidemiology, Jan 1969, Dr. D. G. Ahearn of Georgia State College and Dr. W. Kaplan, Laboratory Division, NCDC, Atlanta, Georgia, reported that while investigating optimal conditions for cold storage of frankfurters, they obtained numerous isolates of *Sporotrichum schenckii.* This is the etiological agent of sporotrichosis, a subacute or chronic disease of man and lower animals. The mycosis is distributed throughout the world and occurs sporadically in both temperate and tropical regions. The disease is generally limited to the skin and subcutaneous tissues. Infections usually develop after the traumatic implantation of fungal propagules from an exogenous source. In rare cases, sporotrichosis may be seen primarily as a systemic disease following pulmonary inception. In such instances it is hypothesized that the agent entered the body by inhalation.

S. schenckii exists in nature as a saprophyte; it has been isolated from soil, water, animal dung, decaying wood, and various plants and plant products. This is the first time *S. schenckii* on cold-stored meat products has been demonstrated.

CHOLERA IN 1968

WHO Wkly Epid Rec 44(17): 285–286, Apr 25, 1969.

Proper surveillance of cholera is one of the most difficult public health problems because of the poor basic health and laboratory services in the cholera-affected countries.

There is no uniform system of reporting cholera cases to WHO. Most health administrations report the number of clinical cases admitted to hospitals, although the clinical features of cholera can be extremely varied. Some report only after bacteriological examination, while others follow up the clinical diagnosis by laboratory confirmation. It is possible to follow the trend of the disease from figures reported by the health administrations. On the basis of reports received up to 17 March 1969, there were 28,941 cases of cholera during 1968. Prior to 1960, the lowest incidence of cholera, 40,110 cases, was recorded in 1955. The fall in incidence after 1964 has continued over the last 4 years. However, in view of the fact that the number of cases in 1968 increased over that for 1967 by about 25%, it can be assumed that this trend of low incidence may not continue.

The increase in the reported total figures for 1968 is more significant considering the extremely low number of cases reported during the year from the Republic of Vietnam. Cases from India and Pakistan constituted 88% of the world total; in India the largest number of cases was reported from Madras, and from Multan in West Pakistan.

Prior to this seventh pandemic, cholera was reported from only 3 to 5 countries, and it is therefore important to note that although there has been no extension of the disease to new territories during 1967 and 1968, it is still being reported from 10 geographical areas, in many of which it has probably become endemic. The reappearance of cholera in Malaysia and Singapore after being quiescent for several years is particularly significant.

There was an outbreak of gastroenteritis in the Sudan in Nov 1968 which initially resembled an outbreak of cholera but was subsequently proved not to be cholera but gastroenteritis due to nonagglutinable vibrios. The outbreak was very promptly investigated and controlled by the government of Sudan, with WHO.

MORTALITY FROM CIRRHOSIS OF THE LIVER AND FROM ALCOHOLISM

WHO Chron 23(5): 243, May 1969.

Over the 10-year period 1956-65, mortality from cirrhosis of the liver increased in Austria, Belgium, Chile, Czechoslovakia, France, Germany (Federal Republic), Greece, Israel, Italy, Puerto Rico, the USA, and West Berlin, according to figures on more than 40 countries and territories published in a recent issue of the WHO *World Health Statistics Report.* In general, however, the median death rates for both sexes remained about 6 per 100,000 population.

The highest death rate from the disease in 1956 was 32.5 per 100,000 population, in France, and the lowest was 1.1, in the United Arab Republic. In 1965, West Berlin had the highest death rate of 44.7 per 100,000 population, followed by France with 34.2 and Portugal with 30.4. The lowest death rates in 1965 were in Iceland, 2.1, and England and Wales, 2.9.

Deaths attributed to cirrhosis of the liver were reported in infants under one year of age in 1965. Death rates increase with age, reaching a peak in the age group 75 years and over. Death rates for males are higher than those for females, and the gap seems to be widening.

The median death rates from alcoholism remained at practically the same level from 1956 to 1965. The highest death rate in 1965 was 12 per 100,000 population and the lowest was 0.1. Deaths from alcoholism start in the 25-34 year age group, and the rates increase with age, reaching a peak in the 65-74 year age group. At all ages the rates for men are higher than those for women.

STREPTOCOCCUS IN THE SALAD

The Lancet I(7608): 1249, June 21, 1969.

Anyone who has had medical care of a closed community of children or young adults knows the usual pattern of an epidemic of streptococcal pharyngitis. Since a sore throat is a symptom seen almost every day, the first 1 or 2 cases in an epidemic seldom give warning of what is to follow. The number of new infections increases slowly and fresh ones may appear over a fortnight or more. Spread is commonest among those who work, play, or sleep together, and a varying proportion of those who acquire the specific organism have only negligible signs of infection. An epidemic in 1968 at the U.S. Air Force Academy, Colorado Springs, Colorado, differed from this usual pattern in many ways. On one day nearly 1,000 of the 3,012 cadets were reported sick with complaints of sore throat, headache, and high fever. The next day another 200 were added to this total. About 100 in all were sick enough to be hospitalized. Within a few hours *Streptococcus pyogenes* (group A) was detected in the throat-swabs by fluorescent microscopy, and within 2 days prophylactic penicillin (or erythromycin for those allergic to penicillin) was given to those who had so far escaped infection; 17% of these showed evidence of infection within the next week. The number of asymptomatic infections was no more than about 2%. Further examination of the streptococcus showed that it belonged to type 12, but no M protein could be found with any available antisera.

Common experience ruled out a single human carrier as the source of such massive simultaneous infection and suggested some article of food or drink. The first wave of infection was distributed fairly evenly over the whole community of cadets (but not among the staff of the Academy) and the dining-hall was the only place where all assembled at the same time. A hint came from the fact that a single class of 61 cadets had been absent on duty from one main meal on the day preceding the outbreak. None of these showed any evidence of infection in the first days of the epidemic, but 5 had symptoms later, presumably because of infection from a secondary source. At the same time 9 out of 28 casual visitors were infected who had eaten only the suspect meal and no other. Food histories taken from a sample of the victims of the epidemic and those who escaped infection suggested that either tuna salad or "cold cuts" might have been the vehicle of infection, and statistical analysis of the results put the blame on the salad beyond reasonable doubt. The salad contained tinned tuna, boiled eggs, mayonnaise, and relish, but of these all but the eggs came from containers which had not been open previously. Ninety-six dozen eggs had been boiled and sliced 24 hours before the salad was made and left at 58° F for 18 hours. The finished salad had been held at 38° F for 6 hours before it was served. Experiment showed that the epidemic strain of *Strep. pyogenes* would multiply 6-fold when inoculated on the surface of hard-boiled eggs held under these conditions, and it is likely that a large mass of eggs would hold the initial heat longer. Throat cultures (nasal cultures might have been more informative) were taken from the 229 members of the dining room staff. Three were found to harbour *Strep. pyogenes* type 12, and 1 of these had been among the 6 men who had prepared the salad. He denied any respiratory symptoms and had no obvious superficial infection.

The disease which occurred among the cadets in the week after the first outbreak—presumably spread from person-to-person—was noticeably milder than that seen earlier. Was the virulence of the streptococcus on the decline or was the dose received smaller? In contrast to the usual American experience no serious complications such as rheumatic fever or nephritis were detected, in spite of a vigorous inquiry.

This interesting story may convince some doubters that diagnosis by fluorescent microscopy—"instant bacteriology"—is more than a parlour trick. Delay in reporting and in identifying the causative organism have been big stumbling blocks to the epidemiologist, and any method which will remove these impediments deserves intensive consideration.

PUBLICATION

The chart, *Emergency Medical Treatment for Acute Pesticide Poisoning,* has been revised by the U.S. Navy Disease Vector Control Center, Naval Air Station, Jacksonville, Florida 32212. Modes of action, toxicity, symptoms and treatment for poisoning by insecticides, rodenticides, herbicides, and solvents are presented. The chart is a convenient size to be posted in emergency treatment rooms and in other locations where it would be advantageous to have the information available for quick reference.

Copies will be distributed to the naval hospitals and dispensaries. Other activities desiring the chart should submit a written request directly to the Officer-in-Charge of the Disease Vector Control Center.

HEPATITIS—TWO OUTBREAKS

USDHEW PHS NCDC Hepatitis Surveil Rep No. 30, Pgs 6–8, Apr 21, 1969.

A. *Los Angeles County, California*

From 28 July through 7 Sept 1968, 75 cases of hepatitis occurred in Harbor Health District, population 156,000, 1 of 23 health districts in Los Angeles County, California. The outbreak was confined to the middle of the upper middle socio-economic area of San Pedro, population approximately 60,000.

Most of the 75 cases were reported to the district health office, the remaining few were discovered by telephoning all internists, general practitioners, and pediatricians in the district and by questioning known cases concerning illnesses in their associates.

Seventy cases were interviewed in person and/or by telephone. Two acquired their infection outside of Los Angeles County and 9 other cases gave histories of contact with a previous case 2-to-6-weeks prior to onset of their illness. The remaining 59 gave no history of known contact with a previous case and denied receipt of blood transfusion and self-injection of parenteral drugs.

Of these 59 cases with no obvious source of infection, 47 were icteric, 12 were anicteric. A diagnosis of anicteric hepatitis was accepted if the person had 3 or more of the following symptoms: anorexia, nausea, abdominal pain, fatigue, light stools, dark urine, and loss of taste for tobacco.

Forty-two (62%) of the cases were 20 years of age or over. The sex distribution was essentially equal with 37 males and 31 females.

The explosive nature of the epidemic curve and the unusual age distribution of the cases led to a search for a common source exposure. Common shopping or attendance at a supermarket, dairy, restaurant, drive-in, etc. could not be established. The municipal water supply seemed an unlikely source. Aside from occasional clusters of friends and family members, the cases in general were socially unacquainted.

Several families recalled eating whipped cream cakes purchased in mid-July from a single bakery, centrally located in the San Pedro business district. Of the 5 families with multiple cases, all gave a history of ingestion of whipped cream cakes purchased from the bakery and eaten approximately 4 weeks prior to the onset of their illness. Of the 59 cases with no history of contact with a known case of hepatitis, 48 cases gave a history of ingestion of whipped cream cakes, prepared by the bakery between 10–26 July 1968. For 41 persons, who recalled a single exposure to whipped cream cakes, their dates of ingestion occurred during the 10-day period 17 to 26 July, with the mean interval between ingestion of whipped cream cakes and onset of illness being 30.2 days, a range of 22–42 days.

The whipped cream cake was a food item popularly served at parties and gatherings. Thirty-six of the ill persons ingested their cakes at 15 such social gatherings. Of 171 total persons investigated, 154 persons admitted to cake ingestion; 36 (23.2%) developed hepatitis. None of the 17 persons at the parties not eating cakes became ill.

Investigation of the bakery revealed no illnesses in July suggestive of hepatitis among the 16 employees. Four employees directly responsible for bare-hand spreading of whipped cream icing on the bakery's products were carefully questioned. It was found that a husband of one of the whipped cream spreaders had experienced an illness quite compatible with anicteric hepatitis. On 10 Aug he became anorectic, fatigued, and noted dark urine. He denied prior ingestion of whipped cream cakes or any other product from the bakery, contact with a known case of hepatitis, and self-injection of parenteral drugs. It is possible that this man contracted his hepatitis in mid-July from his wife, who, while she had a totally asymptomatic but infectious illness, continued to work and contaminated the whipped cream cakes at the bakery.

B. *Nashville, Tennessee*

During the first 11 months of 1968, an increase in the number of hepatitis cases in narcotics users was discovered in Hubbard Hospital, Nashville, Tennes-

see. Preliminary investigations suggested 75 persons actively engaged in narcotics abuse and needle sharing, some of whom were known hepatitis cases.

Of 8 total hepatitis cases admitted to the hospital between 1 Jan and 18 Nov 1968, 7 were narcotics users. The 8th had received a 2-unit blood transfusion 2 months prior to her illness. All 7 narcotics users had onsets of illness between Apr and Nov 1968, were Negroes, 19 to 35 years old, were acquainted with each other, and were part of a much larger group of persons who commonly practiced needle sharing in pursuit of their habit. One of the patients expired of fulminating hepatitis 12 Aug 1968, 4 weeks after his admission to the hospital. His death was followed 9 weeks later, on 19 Oct, by the hospitalization of his girl friend with the same illness. During her hospitalization she confided knowing at least 75 additional persons who commonly practiced needle sharing with her, her deceased boy friend, and several others among the hepatitis patients. Her hospital physicians felt that immune serum globulin should be offered to these contacts, and the patient volunteered that she could bring these persons together for such a purpose. Her statement led to the start of the investigation.

A review both of hepatitis cases reported to the Davidson County Health Department, and hospital admissions between 1 Jan to 15 Nov 1968 at Nashville General, Vanderbilt University Hospital, and Hubbard Hospital, the 3 institutions at which addicts seek medical care, revealed 6 additional cases of drug-associated hepatitis. Three of the 6 were Negroes who were part of the large clique of drug users referred to above. The remaining 3 were Caucasians; 1 was a parenteral amphetamine user who died of acute yellow atrophy 4 days after her admission to Nashville General Hospital. It is noted that of the 5 hepatitis cases reported to the Davidson County Health Department, all were known or suspected drug users and had been admitted to 1 of the 3 hospitals surveyed.

An attempt was made to define the point prevalence of anicteric and icteric illnesses among the large group of Negro drug users who were unknown to the health officials. Despite excellent cooperation from the Hubbard Hospital staff and Meharry Neighborhood Health Center, only 4 patients could be enticed to submit serum for an SGOT determination. Two of the patients, both admitted drug users, had slightly elevated SGOT values of 60. The other 2 were normal.

Editorial Comment: The extent of the problem of drug-associated hepatitis among parenteral narcotics abusers who commonly share needles was only touched upon by this investigation. The public health importance of hepatitis among addicts has its greatest impact on recipients of the whole blood which addicts often sell to support their habit. While this reminder may be often repeated, the 70-fold greater risk involving blood obtained from addict donors versus healthy donors, in transmitting hepatitis to recipients, still needs to be emphasized.

KNOW YOUR WORLD

Did You Know?

That in the United States 48 cases of paralytic poliomyelitis were reported in 1968, a slight increase over the 40 cases in 1967?

This overall rise is clearly attributable to an increase from 9 to 20 poliomyelitis cases in Texas. The number of non-Texas cases has fallen steadily from 37 in 1966 and 31 in 1967 to 28 in 1968. The 1968 cases occurred in many parts of the country, although there were relative concentrations along the Texas-Mexican border and in the Midwest. Most 1968 cases were unimmunized infants and preschool children of lower socioeconomic background. Type 1 poliovirus was again the dominant serotype. The 41 case total reported for 1967 included 1 delayed report from 1966. The totals for 1966 and 1967 have now been revised accordingly, and shall hereafter be listed as 103 for 1966 and 40 for 1967.[1]

That the total of 2,610 cases of malaria reported in the United States in 1968 was slightly less than the 1967 total of 2,855?

Vietnam returnees continued to account for over 90% of the cases. *P. vivax* accounted for 81% of the infections and *P. falciparum* 13%. Twelve cases were acquired in the U.S. Five of these, all due to *P. vivax,* were introduced cases; and 7, 3 due to *P. faciparum,* 3 due to *P. malariae,* and 1 due to *P. vivax,* were induced cases. Of the introduced cases, 4 occurred in civilians and were epidemio-

logically related. There were 6 malaria fatalities, all *P. falciparum* infections: 2 in seamen, 2 in Vietnam returnees, 1 in an American tourist, and 1 in the recipient of a blood transfusion.[2]

That from 1963 through 1968 measles immunization is estimated to have averted 9.7 million acute cases and 3,244 cases of mental retardation?

The immunization effort against measles in the United States was initiated in 1963 and has resulted in a sharp decrease in incidence of the disease—from 4 million cases in 1963 to ¼ of a million cases in 1968—and in associated costs.

It also is estimated to have saved 973 lives, 555,000 hospital days, 291,000 years of normal life, more than 1.6 million workdays, 32 million schooldays, and $423 million. About 9/10 of the savings in each of these categories has been realized in the last 3 years—the period of intensive national effort to eradicate measles.[3]

That there are about 5 million asthmatic youngsters in the U.S.?

More than 50% of children in a marriage in which 1 partner has a history of allergy will have one of the major allergic illnesses—asthma, eczema, hay fever or hives—during the first 6 years of life. About ⅓ of allergic people have infantile eczema, and between ½ and ¾ of all infants with infantile eczema ultimately develop respiratory allergy. Prevention of asthma starts in infancy.[4]

That people in the canned seafood industry and at the Food and Drug Administration receive complaints from consumers who believe they have found glass in their canned seafood—especially shrimp?

Examination reveals it is not glass at all, but "struvite" crystals—crystalline magnesium ammonium phosphate to the chemist—which occasionally forms in canned seafood from normally present constituents. While struvite is not desirable, it does not affect the safety of the food at all, according to FDA. Seafood canners have devoted considerable attention to the problem of struvite formation, but they still are not able to prevent it. If you do happen to find a hard, clear crystalline bit of material in canned seafood, a simple test to distinguish between struvite and glass is simply to place the material in warm vinegar for awhile. The struvite crystals will dissolve; glass of course, will not.[5]

That schistosomiasis was recently diagnosed in 5 members of a family in Decatur, Georgia?

They had lived in Uganda for several years and had swum in fresh water lakes and streams there. They returned to the United States in 1967. In January 1969, the father was discovered to harbour numerous viable *Schistosoma mansoni* eggs. The mother and 3 children were asymptomatic, but because of the possibility of a common exposure to schistosome cercariae while in Africa, skin, serologic tests and stool examinations were done on all family members. All had *S. mansoni* eggs in their stools, positive skin tests, and at least 1 positive serologic test—some had more than one. The father was treated with sodium antimony dimercaptosuccinate (Astimaban—available through the Parasitic Disease Drug Service, NCDC). The mother and 3 children will be treated with other schistosomicidal agents.[6]

Although there has been a gradual decline in the reported incidence of trichinosis in the U.S. in the past 2 decades, 84 cases of trichinosis were reported in 1968 compared to 67 for 1967?

The increased incidence might be attributed to the intensified surveillance begun in 1967. In 1968 there were 5 outbreaks of 4 or more cases, and the first trichinosis-related death in 2 years was reported. The majority of infections were acquired from commercially processed pork products consumed at home. Farm-raised and processed swine accounted for 4 cases and 1 death while 10 cases in 2 outbreaks were due to wild game.[7]

That births in the U.S. totaled 3.47 million during 1968, the lowest number since 1946?

The number of births in 1968 was slightly more than 1% below the 1967 total. This was the smallest annual decline since 1964. Total births were 19% below the record total of 1957.

The 1968 birth rate—17.4 births per 1,000 total population—continued to drop from the 1957 peak of 25.3, and set a new low figure for the United States.[8]

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EDITOR'S SECTION

WITHDRAWAL OF OFFICER RESIGNATIONS

The following information was released in "The Navy Personnel Newsletter" dated 24 July 1969 from the Chief of Navy Personnel.

"During the continuous review of ways and means to improve officer retention, it has become evident that many officers who have submitted their resignations now desire to withdraw them. Many officers are reluctant to do so in the belief that the cancelled resignation on file in their official records would prove prejudicial to their interests in the future.

Although such fears cannot be substantiated, the Secretary of the Navy has approved a radical departure from previous policy in this regard. When the withdrawal of a resignation is approved, *and the officer has specifically so requested in his withdrawal request,* all resignation-related correspondence will be removed from the record and returned to the officer concerned."

CHEMICAL, BIOLOGICAL AND RADIOLOGICAL WEAPONS ORIENTATION COURSE

The Chemical, Biological, Radiological Weapons Orientation course will be conducted by the U.S. Army at the Dugway Proving Ground, Dugway, Utah, during fiscal year 1970. The duration of the course is three and one-half days.

Officers of the rank of Lieutenant Commander or above are eligible to attend. Civilians in the grade of GS-12 or higher must be in a key position where need-to-know is mandatory. Officers of the rank of Lieutenant and civilians in the grade of GS-11 may be granted waivers where special circumstances warrant their attending the course. All requests for waivers must be accompanied by job description and need-to-know certification. Persons who have received complete CBR briefings during the past two years should consider delaying their attendance. Security clearance of INTERIM TOP SECRET is required. Limited quotas will be provided the Bureau of Medicine and Surgery by the Chief of Naval Personnel on a "first come first serve" basis. Requests should be forwarded in accordance with BUMEDINST 1520.8 Series.

The course provides a high level orientation on Chemical and Biological Warfare, and Radiological Implications of Nuclear Warfare, and is designed to acquaint military and civilian personnel of the Armed Forces with United States doctrine, policy, techniques and capabilities in CBR Warfare.

Convening Dates of Courses

27 Oct 1969	16 Mar 1970
3 Nov 1969	30 Mar 1970
17 Nov 1969	13 Apr 1970
1 Dec 1969	20 Apr 1970
8 Dec 1969	27 Apr 1970
23 Feb 1970	18 May 1970
2 Mar 1970	25 May 1970
9 Mar 1970	1 Jun 1970

—Training Div, BuMed.

HUMAN HISTOCOMPATIBILITY TYPING CENTER

A major new Human Histocompatibility Typing Center, established by the NIH National Cancer Institute to facilitate transplantation of organs and matched blood components to cancer patients, recently began operations.

The center analyzes tissue samples and computer-stores information on tissue types so that it will be possible to locate compatible donors whenever NCI patients require white blood cell or blood platelet transfusions, or bone marrow transplants. By facilitating the testing of cell fractions, the center will also open the way to large-scale preparation of special proteins called transplantation antigens which, in the future, may be administered to permit effective transplantation of unmatched tissue and organs.

Under a contract awarded to Microbiological Associates, Inc., a biomedical research firm in Bethesda, Maryland, the center will conduct cell typing tests similar in concept to blood typing tests. The tests will identify transplantation antigens present in an individual's tissue. It is the presence of foreign transplantation antigens on transplanted tissue that causes the rejection of donor organs and tissues.

NCI project directors, Drs. John L. Fahey, Ronald Yankee, and G. Nicholas Rogentine estimate that 50-60 tissue samples from NCI patients, their relatives, and NIH Blood Bank donors will be analyzed each week. Under the direction of Dr. Robert John Trapani of Microbiological Associates, the new laboratory will be able, within hours, to type a patient's tissue and locate donors with a compatible type for tissue transplantation.

White blood cell transfusions increase resistance to infection, and platelet transfusions elevate low platelet counts and help stop bleeding in patients with aplastic anemia and acute leukemia. Bone marrow transplants will be used to replace the marrow of acute leukemia patients who do not respond to conventional therapy, and of other cancer patients whose marrow is depleted as a result of intensive drug treatment.

Immune rejection of grafted tissue remains a central problem to transplantation immunology. Although tissue typing is a valuable method of matching donors and recipients and helps prevent rejection by reducing the immunological differences between the two, its application is limited at the present time because compatible donors are difficult to find.

Some scientists believe that an ideal way to suppress transplant rejection would be to use soluble transplantation antigens to induce specific tolerance. Preliminary studies in animals indicate that if foreign transplantation antigens are administered in low doses (a procedure somewhat similar to allergy immunizations) the body's immune mechanism will become tolerant to them and accept a transplant from an individual with those antigens.

It is anticipated that these and other advances in the immunotherapy of human cancer will be furthered by research at the new Center, supported this year by a NIH contract for $126,400 administered by the National Cancer Institute.—NIH, National Cancer Institute, Bethesda, Md.

NAVY UNIT COMMENDATION

NSA Hospital, DaNang, as a component of the Naval Support Activity, DaNang has been awarded the Navy Unit Commendation on two occasions, the first for service from 15 October 1965—15 August 1966 and the second award for the period 16 August 1966 to 15 May 1969. Many other Navy Medical Department personnel have earned the Presidential Unit Citation, Navy Unit Commendation and the Meritorious Unit Commendation while serving in Navy and Marine Corps Units in Vietnam and its adjacent waters.

AHA MEETING

The Navy was well represented at the American Hospital Association's 71st annual meeting in Chicago, Illinois, August 17-21, 1969. Six Medical Service Corps officers were elevated from nominee status to membership in the American College of Hospital Administrators: Captains Emmett L. Van Landingham, Jr., and Robert M. Tennille, Jr.; LCDR Francis J. Redding; Lieutenants David E. Bohner, Robert P. Kowal and Christopher M. Warner. CAPT Van Landingham is Chief of the Medical Service Corps; CAPT Tennille is CO of the Naval School of Hospital Administration; LCDR Redding is a faculty member at NSHA; LT's Bohner and Kowal are on duty at the Navy Hospital, Guam, M. I. LT Warner is on duty at Oakland Naval Hospital. Two other MSC officers, CDR J. P. Kirsch and LTJG R. F. Eckroyd were admitted to nomineeship at the meeting. VADM Robert B. Brown, MC USN (Ret) was honored by the Executives of the Federal Hospitals on August 19, receiving a Certificate of Appreciation signed by the heads of all the Federal Medical Agencies. VADM George M. Davis, Surgeon General, presented the certificate which lauded a "long and distinguished career", noting that VADM Brown "was wholly dedicated to the noblest standards of professional medicine and earned for himself the respect and admiration of the entire medical community". VADM Brown, former Surgeon General, retired this year after a 27 year career in the Medical Corps of the U.S. Navy.

CAPT Roger Stevenson, MC USN, Director of Professional Division, Bureau of Medicine and Surgery, took part in a panel of speakers representing the Federal Medical Services. CAPT Stevenson spoke for the Navy on "Federal Experience in Extension of Professional Services in the Use of Trained Health Care Assistants". The text of his presentation appears on page 1.

LCDR Phyllis J. Elsass, NC USN, monitored the Navy Medical Department segment of the Federal Hospitals' Exhibit ably assisted by HMC Warren Williams, Jr., and Miss Irene Sedlak. The popular project was entitled "Research in the Delivery of Health Services" and focused on Ward Manager Research conducted by the Nursing Research Division of the Naval Medical Research Institute at NNMC, Bethesda, Md. The Exhibit was attractively constructed and managed by personnel of the Naval Medical School and Armed Forces Institute of Pathology. Great interest in the Ward Manager concept was shown by administrative personnel and representatives of educational institutions. A number of physicians also inquired into the program and remarked on the implications of ward managers and other paramedical personnel for civilian nursing and medical practice.

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